

EXHIBIT B

**UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION**

IN RE NATIONAL COLLEGIATE ATHLETIC ASSOCIATION STUDENT- ATHLETE CONCUSSION INJURY LITIGATION

MDL No. 2492

Master Docket No. 1:13-cv-09116

This Document Relates To:
All Cases

Judge John Z. Lee

Magistrate Judge Geraldine Soat Brown

DECLARATION OF DR. JULIAN E. BAILES

I, Dr. Julian E. Bailes, declare as follows:

1. I am the Chairman of the Department of Neurosurgery and Co-Director of the NorthShore Neurological Institute in Chicago, Illinois and a Clinical Professor of Neurosurgery at the University of Chicago Pritzker School of Medicine.

2. I am a licensed physician in the states of Illinois, Pennsylvania, and West Virginia, as well as a board-certified neurological surgeon who has been either a division chief or departmental chairman for my entire career.

3. I have authored approximately 300 peer-reviewed publications, abstracts, and book chapters, written five books, and given nearly 500 presentations at national and international medical conferences. I also have editorial duties, serving for the last five years as Editor of Sports and Rehabilitation of the journal Neurosurgery, and I am a member of the editorial board of several journals, including the Journal of Neurotrauma.

4. Since 2002, I have been the Chairman of Sports Medicine for organized neurosurgery's two organizations, the American Association of Neurological Surgeons and the Congress of Neurological Surgeons. I have been a sideline and team physician at either the

National Football League (“NFL”) or the National Collegiate Athletic Association (“NCAA”) levels for the last 25 years. I am also the Chairman of the Pop Warner Football Medical Advisory Committee, which represents the oldest and largest youth football league in the United States.

5. I have received over \$27 million in research funding and maintain a basic research laboratory and supervise a research team that investigates experimental brain injury, its causes and prevention. I am the founding member and Co-Director of the Brain Injury Research Institute, which deals with issues related to the causes of chronic brain injury in sports and the military. A copy of my curriculum vitae is attached as Exhibit 1.

6. In 2001, Dr. Kevin Guskiewicz and I established the Center for the Study of Retired Athletes at the University of North Carolina, Chapel Hill, where I remain the Medical Director. My interest in the long-term effects of repetitive brain injury and cranial impacts goes back to the late 1990s, when I began, at the request of the NFL Players’ Association, to investigate the health of former professional football players, publishing data for the first time about chronic deleterious brain effects of years of football play. That work resulted in important findings, and I was a co-author of publications in 2005 and 2007 showing the incidence of cognitive impairment and depression in those retired NFL players who had three or more significant concussions.^{1,2}

7. Subsequently, I began a collaboration with Dr. Bennet Omalu, the discoverer of Chronic Traumatic Encephalopathy (“CTE”), through which we have studied, through autopsy

¹ Kevin M. Guskiewicz et al., Association Between Recurrent Concussion and Late-Life Cognitive Impairment in Retired Professional Football Players, 57 NEUROSURGERY 719, 719-26 (2005).

² Kevin M. Guskiewicz et al., Recurrent Concussion and Risk of Depression in Retired Professional Football Players, 39 MED. & SCI. IN SPORTS & EXERCISE 903, 903-09 (2007).

analysis, the brains of over 40 former football players, boxers, and military veterans with CTE.³ We reported the first case of CTE in a military veteran who had been exposed to brain trauma from exposure to multiple blast injuries.⁴

8. Currently, I have been involved with the identification of CTE in living individuals, the first time that this has been shown to be possible, through positron emission tomography (“PET”) scans, in conjunction with my colleagues at UCLA.⁵

9. I have reviewed the Complaint filed in Nichols v. NCAA, No. 1:14-cv-00962 (N.D. Ill.) (Dkt. #1), and Nichols’ Second Objections to Plaintiffs’ Motion for Preliminary Approval of Class Settlement (Dkt. #201). I have also reviewed the concussion guidelines included in the NCAA Sports Medicine Handbook from 1994 to present.

CONCUSSION STANDARDS OF CARE

10. Contrary to Mr. Nichols’s claim, there was no consensus in 2002 regarding concussion management. There was not, in fact, agreement between most major sports medicine organizations and bodies concerning the various aspects of concussion management -- including same-day return to play. Instead, our understanding of sports concussion was just beginning to expand by 2002, and it was nearly another decade before there was a more general concurrence and uniformity in player concussion management.

³ See, e.g., Bennet Omalu et al., Emerging Histomorphologic Phenotypes of Chronic Traumatic Encephalopathy in American Athletes, 69 NEUROSURGERY 173, 173-83 (2011); Bennet Omalu et al., Chronic Traumatic Encephalopathy in a Iraqi War Veteran with Posttraumatic Stress Disorder who Committed Suicide, 31 NEUROSURGICAL FOCUS, Nov. 2011, at 1-10.

⁴ See Bennet Omalu et al., Chronic Traumatic Encephalopathy in a Iraqi War Veteran With Posttraumatic Stress Disorder Who Committed Suicide, 31 NEUROSURGICAL FOCUS, Nov. 2011, at 1-10.

⁵ Gary W. Small et al., PET Scanning of Brain Tau in Retired National Football League Players: Preliminary Findings, 21:2 AM. J. GERIATRIC PSYCHIATRY 138, 138-44 (2013).

11. From 2000 to at least 2010, there was disagreement among the medical and sports communities as to which sideline evaluation policy should be utilized in acute, game-day triage, the role of neuropsychological testing either in baseline format or for the injured player, the utility of radiological imaging studies, the various grading systems and their application, the use of balance testing, and return to play philosophy and methodology, among other things. The assertions of Mr. Nichols and his counsel, however, both misrepresent and grossly oversimplify the evolution of concussion management during this time. Indeed, our understanding of concussions and the best methods for diagnosing and treating concussion continues to evolve today.

12. As an initial matter, it is important to note that there is not a central reporting agency or governmental body that collects data, disseminates emerging research, or establishes policy for the management of the injured athlete. The applicable standard of care for any injury is not pronounced by an organization, such as a trainers' association. Rather, through years of research, experience, and medical evolution in best practices as applied to emerging medical science, a general understanding among practitioners emerges. For example, the 2004 position statement of the National Athletic Trainers' Association ("NATA"), which Mr. Nichols cites as reflecting the standard of care in 2002, was never meant to be, nor did it become, the customary and accepted standard for best practices. Instead, the position statement was exactly that -- a document that reflected merely the opinions of the organization to serve as a guide for its members. At the same time that NATA was publishing its 2004 position statement, however, every major medical specialty that dealt with sports concussion was attempting to define, adopt and promote its own safe and effective policies.

13. Since the 1980s, various organizations have published concussion guidelines. Those guidelines have varied markedly in their approach to concussion treatment and return-to-play. For example, guidelines that Dr. Robert Cantu published in 1986 allowed for same-day return to play in certain instances.⁶ Colorado Medical Society guidelines published in 1990 and 1991 also provided for return-to-play after a “mild concussion” within only twenty minutes.⁷ Six years later, in 1997, I participated in a writing group of experts coordinated by the American Academy of Neurology. This work resulted in another recommended protocol, in which the published concussion management guidelines allowed for same-day return to play for athletes who suffered a “Grade 1” concussion.⁸

14. Between 2000 and 2010, there were many advances in our understanding of the brain’s response to concussive injury, in the biological sense as well as the behavioral expression and clinical course. This resulted from numerous research centers and teams concentrating on the nature of this injury as well as expanded funding opportunities to conduct such research. As our knowledge and momentum in comprehension of sports concussion increased, this led to changes in recommendations for player management. This continued evolution in the science will likely lead to modification of concussion management protocols in the future as well. Specifically, the scientific community’s understanding of the basic metabolic changes, cellular disturbances, and ultrastructural defects of the brain began to emerge and were tied into more common theories on brain injury. Numerous studies, both from the research laboratory and the

⁶ Robert C. Cantu, Guidelines for Return to Contact Sports After a Cerebral Concussion, 14(10) PHYSICIAN & SPORTS MED. 75, 79 (1986).

⁷ Sports Med. Comm., Colo. Med. Soc’y, Guidelines for the Management of Concussion in Sports, 1990 (rev. May 1991).

⁸ Quality Standards Subcomm., Am. Acad. of Neurology, Practice Parameter: The Management of Concussion in Sports (Summary Statement), 48 NEUROLOGY 581, 583 (1997).

clinical realm, defined the various aspects of injury to the neuron, brain supporting cells, the connecting fibers, intracellular organelles, and vascular supply. Two theories emerged that described concussion as occurring on either a metabolic basis or as a disturbance in the vascular distribution to areas of the brain. In response to this emerging science, protocols for concussion management among athletes evolved and were implemented.

15. In 2001, the First International Conference on Concussion in Sport was held in Vienna (the “Vienna Convention.”). The Vienna Convention authors did not purport to impose a one-size-fits-all required protocol for concussion management, but to “provide recommendations for the improvement of safety and health of athletes who suffer concussive injuries in ice hockey, football (soccer), and other sports.”⁹ Importantly, recognizing the evolving nature of the science, the authors also expressly stated that “[t]his protocol represents a work in progress, and, as with all other guidelines or proposals, it must undergo revision as new information is added to the current literature and understanding of this injury.”¹⁰ The authors declined to endorse any particular concussion grading scales, instead stating “[i]n the absence of scientifically validated return to play guidelines, a clinical construct is recommended using an assessment of injury recovery and graded return to play.”¹¹ This means that concussion is at times a subjective diagnosis. It has great variability in its expression, and it must be approached on an individual basis. The authors emphasized that “the science of concussion is at the early stages and therefore

⁹ M. Aubry et al., Summary and Agreement Statement of the First International Conference on Concussion in Sport, Vienna 2001, 36 BRIT. J. SPORTS MED. 6, 6 (2002).

¹⁰ Id.

¹¹ Id. at 7.

management and return to play decisions remain largely in the realm of clinical judgment on an individual basis.”¹²

16. The Second International Conference on Concussion in Sport was held in Prague in 2004. The second conference was established by the same organizations that held the Vienna conference, the Federation Internationale de Football Association, the International Olympic Committee, and the International Ice Hockey Federation. Because there had not been any significant scientific or medical breakthroughs since the first meeting, the participants addressed all basic sports concussion topics.¹³

17. In 2004, NATA published its own set of concussion management guidelines. Mr. Nichols misstates the content of these guidelines. First, he wrongly asserts that they were part of a supposed “consensus” reached in 2002 when in fact they were not published until 2004.¹⁴ In addition, the NATA guidelines do not contain specific “stepwise” return to play guidelines, each step of which takes a minimum of one day. Mr. Nichols also incorrectly asserts that the NATA guidelines included “specific return-to-play guidelines” under which a player should never be allowed to return to play the same day he or she sustains a concussion. In fact, the NATA guidelines clearly contemplate that some players may return to play the same day, noting that “[m]ore recent studies of high school and collegiate athletes underscore *the importance of ensuring that the athlete is symptom free before returning to participation on the same day.*”¹⁵ “*Athletes who return on the same day because symptoms resolved quickly*

¹² *Id.* at 9.

¹³ P. McCrory et al., Summary and Agreement Statement of the 2nd International Conference on Concussion in Sport, Prague 2004, 39 BRIT. J. SPORTS MED. 196, 196-204 (2005).

¹⁴ Kevin M. Guskiewicz et al., National Athletic Trainers’ Association Position Statement: Management of Sport-Related Concussion, 39(3) J. OF ATHLETIC TRAINING 280, 280-97 (2004).

¹⁵ *Id.* at 290 (emphasis added).

(<20 minutes) should be monitored closely after they return to play.”¹⁶ Notably, Dr. Cantu, who Mr. Nichols cites in his objections, was listed as an author on that report, undermining any assertions that there was a “no same-day return to play” consensus as early as 2002 (or even 2004).

18. While Mr. Nichols attempts to portray the 2004 NATA guidelines as reflecting a broadly-accepted consensus, a 2005 publication by leaders in NATA concluded that “only a small percentage of certified athletic trainers currently follow the guidelines proposed by [NATA]. Various assessment methods and tools are currently being used, but clinicians must continue to implement a combination of methods and tools in order to comply with the position statement.”¹⁷ This emphasized the lack of uniformity, agreement, and standards in practice.

19. The lack of a definitive “consensus” is further reflected in a 2006 article, in which Dr. Cantu discussed the numerous concussion conferences held between 2000 and 2006 and stated:

This subject is perhaps best summarized by the final comment in the 2004 Prague summary, which states, “this protocol represents a work in progress, and, as with all other recommendations or proposals, it must be updated as new information is added to the current state of the literature and understanding of this injury.”¹⁸

20. The third and fourth International Conferences on Concussion in Sport were held in Zurich in 2008 and 2012.^{19,20} During these meetings, myriad concussion issues were

¹⁶ Id. at 282 (emphasis added).

¹⁷ Andrew J. Notebaert & Kevin M. Guskiewicz, Current Trends in Athletic Training Practice for Concussion Assessment and Management, 40(4) J. OF ATHLETIC TRAINING 320, 320 (2005).

¹⁸ Robert C. Cantu, An Overview of Concussion Consensus Statements Since 2000, 21(4) NEUROSURGICAL FOCUS, Oct. 2006, at 4.

¹⁹ See P. McCrory et al., Consensus Statement on Concussion in Sport: the 3rd International Conference on Concussion in Sport held in Zurich, November 2008, 43(Suppl. I) BRIT. J. SPORTS MED. i76 (2009).

addressed, and writing committees produced documents which reflected those discussions.

Those documents include summaries of: (i) updates of emerging scientific thought and discovery; (ii) advances in protective equipment, concussion management and prevention; and (iii) developments in concussion science. The authors of all of these statements acknowledged that the science of concussion was still evolving and remained a work in progress, and that management and return-to-play decisions should remain in the realm of clinical judgment and on an individualized basis.

21. For almost the entire decade from 2000-2010, the NFL, National High School Federation, Pop Warner Football, USA Football, the American College of Sports Medicine, as well as national organizations in other sports such as ice hockey, soccer, lacrosse and others, the American Association of Neurological Surgeons, the Congress of Neurological Surgeons, and the American Academy of Neurology had no universal or mandatory policies concerning concussion management.

22. As additional research improves the scientific community's understanding of concussions, concussion management practices continue to evolve. In 2013, several new or updated clinical practice guidelines and position statements were published on the diagnosis, treatment and management of mild traumatic brain injury and concussion in sports. Three of these guidelines were produced by the American Medical Society for Sports Medicine, the American Academy of Neurology, and the Zurich Consensus working group. The goal of each group was to clearly define best practices for the definition, diagnosis, and acute and post-acute management of sports-related concussion, including specific recommendations for return to play.

(...continued)

²⁰ See Paul McCrory et al., Consensus Statement on Concussion in Sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012, 47 BRIT. J. SPORTS MED. 250 (2013).

All three emphasized the continued ambiguity and lack of agreement in various aspects of concussion management. For example, the American Medical Society's position statement acknowledged that "[a]dditional research is needed to validate current assessment tools, delineate the role of [neuropsychological] testing and improve identification of those at risk of prolonged post-concussive symptoms or other long-term complications. Evolving technologies for the diagnosis of concussion, such as newer neuroimaging techniques or biological markers, may provide new insights into the evaluation and management of sports concussion."²¹ Similarly, the 2012 Zurich Consensus working group stated that "[w]hile agreement exists pertaining to principal messages conveyed within this document, the authors acknowledge that the science of concussion is evolving, and therefore management and return to play (RTP) decisions remain in the realm of clinical judgment on an individualised basis."²²

23. Accordingly, Mr. Nichols' contention that in 2002 a consensus on concussion management and treatment had been accepted is simply belied by the facts. We in the scientific community recognize that there is still much for us to learn about how the brain responds to injury and how best to prevent and treat such injuries.

**NCAA GUIDELINES WERE CONSISTENT
WITH EVOLVING STANDARDS OF CARE**

24. The NCAA materials I have reviewed and am familiar with demonstrate that the NCAA adhered to and adapted the emerging understanding for concussion management. In particular, the NCAA has for almost 20 years recommended that student athletes not be

²¹ Kimberly G. Harmon et al., American Medical Society for Sports Medicine Position Statement: Concussion in Sport, 47 BRIT. J. SPORTS MED. 15, 16 (2013).

²² See McCrory, supra note 20, at 250.

permitted to return to play while exhibiting concussion symptoms. As new and alternative views developed, the NCAA updated its guidance.

25. For example, in 1994, the NCAA added a section to its Sports Medicine Handbook called “Concussion and Second-Impact Syndrome,” which incorporated the 1990 Colorado Medical Society guidelines.²³ These guidelines required that an athlete who suffers a grade-3 concussion should never return to play on the same day.²⁴ The handbook also noted that “[a]lthough these guidelines may assist in clinical decision-making, *they are not absolute and should not be substituted for the clinical judgment of the examining physician.*”²⁵ The NCAA also stressed that “[t]he attending medical staff *should not allow a player to resume participation in sports until the injured student-athlete has fully recovered from his/her postconcussive symptoms.*”²⁶

26. In 1997 (after the publication of the American Academy of Neurology guidelines), the NCAA revised the “Concussion and Second-Impact Syndrome” section in its Sports Medicine Handbook. While the NCAA continued to stress that a symptomatic player should not return to play, it also noted the growing lack of consensus over concussion treatment and care and emphasized that “[a]lthough the grading scales and return-to-play criteria currently in the literature may assist in the clinical decision-making for the student-athlete who has suffered a concussion, these grading scales and return-to-play criteria should not be substituted for the clinical judgment of the examining physician.”²⁷ This was four years before the Vienna

²³ 1994-1995 NCAA Sports Medicine Handbook, attached hereto as Exhibit 2, at 40-43.

²⁴ *Id.* at 40.

²⁵ *Id.* (emphasis added).

²⁶ *Id.* at 43 (emphasis added).

²⁷ 1997-1998 NCAA Sports Medicine Handbook, attached hereto as Exhibit 3, at 44.

Convention participants recommended abandoning strict adherence to concussion grading scales in favor of individualized assessments.

27. The NCAA's published guidelines were fully consistent with the recommendations of the Vienna Protocol and the NATA guidelines Mr. Nichols cites. For example, the NCAA cautioned against relying on previously promulgated return-to-play guidelines and grading scales, emphasizing the individualized nature of return-to-play determinations.²⁸ The NCAA also made clear that no athlete should return to play while symptomatic.²⁹

28. As early as 1997, the NCAA's guidelines contained the warning that "[a] student-athlete rendered unconscious for any period of time should not be permitted to return to the practice or game in which the head injury occurred. In addition, no student-athlete should be allowed to return to athletics activity while symptomatic."³⁰

29. In 2004, the NCAA amended its Sports Medicine Handbook, in part to note that "recent publications have endorsed the use of neurocognitive or neuropsychological testing" but that "[f]urther research is needed to understand the complete role of neuropsychological testing."³¹ The NCAA also added recommendations for step-wise return to play, and emphasized that "[i]t is essential that no athlete be allowed to return to participation when any

²⁸ Compare M. Aubry et al., Summary and Agreement Statement of the First International Conference on Concussion in Sport, Vienna 2001, 36 BRIT. J. SPORTS MED. 6, at 6-10 (2002) (noting that there is an "absence of scientifically validated return to play guidelines" and stating that "decisions on return to play" should be made "individually"), with 2001-2002 NCAA Sports Medicine Handbook, attached hereto as Exhibit 4, at 52-53 (recognizing that "[s]everal grading scales have been proposed" but that "these categorizations vary and are not universally accepted" and "should not be substituted for the clinical judgment of the examining physician").

²⁹ 2001-2002 Sports Medicine Handbook, Ex. 4, at 52.

³⁰ 1997-1998 NCAA Sports Medicine Handbook, Ex. 3, at 43 (emphasis in original); see also 2002-2003 NCAA Sports Medicine Handbook, attached hereto as Exhibit 5, at 54 (same).

³¹ 2004-2005 NCAA Sports Medicine Handbook, attached hereto as Exhibit 6, at 48.

symptoms, including mild headache, persist.”³² The NCAA again amended its Sports Medicine Handbook in 2009 to include information from the Zurich conference and references to the CDC’s Heads Up: Concussion Tool Kit and Heads Up Video.³³

30. And in 2010-2011, in addition to enacting its Concussion Management Plan legislation which prohibited same-day return to play, the NCAA again re-vamped its handbook to include concussion statistics, more detailed return to play guidelines, and recommended “best practices” for concussion management plans.³⁴ As always, the NCAA emphasized that “it is essential that the medical care team treating athletes continue to rely on its clinical skills in evaluating the head-injured athlete to the best of its ability” and “it is essential that no athlete be allowed to return to participation when any symptoms persist.”³⁵

31. In sum, the NCAA Sports Medicine Handbook gave guidance and recommendations that were consistent with general medical opinion. If during this period, NCAA student-athletes had been treated by the Sports Medicine Handbook policy, they would have received care which was consistent with the standard of care.

**THE VAST MAJORITY OF CONCUSSIONS RESOLVE
COMPLETELY WITH NO LONG-TERM HEALTH EFFECTS**

32. The vast majority of concussions self-resolve in seven days and all but approximately 1% resolve within one year and result in no long-term effects.

33. A diagnosis of concussion does not indicate whether the concussed individual will be among the very small number of people whose symptoms will not completely resolve. Any long-term condition such as post-concussion syndrome (“PCS”) would have to be diagnosed by a

³² Id.

³³ See 2009-2010 NCAA Sports Medicine Handbook, attached hereto as Exhibit 7, at 55.

³⁴ 2010-2011 NCAA Sports Medicine Handbook, attached hereto as Exhibit 8, at 52-56.

³⁵ Id. at 54.

healthcare practitioner on a confidential basis after the concussed individual sought medical care. Such a diagnosis would remain confidential unless the patient waived confidentiality and made public the diagnosis, which very few patients do.

34. Mr. Nichols overstates the extent to which there is agreement within the scientific community that concussions and subsequent concussive or sub-concussive impacts can cause long-term medical conditions, such as chronic neurocognitive impairment, CTE, amyotrophic lateral sclerosis (“ALS”), Parkinsonism, Alzheimer’s disease, and dementia. While researchers are investigating whether causal connections exist between concussions and these conditions, the science is still evolving. The incidence, prevalence, risk and mechanisms by which such conditions occur are not fully understood and remain the source of considerable debate.

35. The most significant cause of chronic neurocognitive impairment is dementia, which has several different types and several different causes. Chronic neurocognitive impairment is a disease of the elderly, and the vast majority of people diagnosed with chronic neurocognitive impairment are diagnosed after age 65.

36. There are many types of dementia, including Alzheimer’s disease, vascular dementia, diabetes dementia, and Pick’s disease. Dementia can have many causes, including genetics, stroke and diabetes. Further, the cause of many types of dementia is unknown. Dementia is a disease of the elderly, and the vast majority of people diagnosed with dementia are diagnosed after age 65.

37. CTE is believed to be caused by exposure to multiple concussive and/or subconcussive impacts. CTE has only been diagnosed in nine people who played a sport in

college but did not go on to play after college.³⁶ Those individuals were diagnosed pathologically. While research is ongoing to identify CTE in living people, currently a definitive diagnosis is only made by autopsy.

38. The causes of ALS are unknown, but certain gene variations appear to increase the risk of ALS.³⁷ ALS is also a disease of the elderly, and the mean age of onset is 65.³⁸ ALS reaches its peak incidence rate for individuals between the ages of 60 and 79.³⁹ Approximately 10-15/100,000 people in this age range are diagnosed with ALS.⁴⁰ ALS has an extremely low incidence in people 40 years old and younger, with an incidence of 1.5/100,000 per year.⁴¹

39. The causes of Parkinsonism are unknown, but some researchers believe it results from genetic mutations or interactions between genetic and environmental factors.⁴² Parkinson's disease is also a disease of the elderly. It has an extremely low incidence in people under age 40, with an incidence of 0.5/100,000 per year.⁴³

³⁶ Joseph C. Maroon et al., Chronic Traumatic Encephalopathy in Contact Sports: A Systematic Review of All Reported Pathological Cases, 10(2) PLOS ONE, Feb. 11, 2015, at 1, 4.

³⁷ See, e.g., Bart Swinnen & Wim Robberecht, The Phenotypic Variability of Amyotrophic Lateral Sclerosis, 10 NATURE REV. NEUROLOGY 661, 662-63 (2014).

³⁸ M. Sabatelli et al., Clinical and Genetic Heterogeneity of Amyotrophic Lateral Sclerosis, 83 CLINICAL GENETICS 408, 408 (2013).

³⁹ Id.

⁴⁰ Id.

⁴¹ Id.

⁴² Tamara Pringsheim et al., The Prevalence of Parkinson's Disease: A Systematic Review and Meta-Analysis, 29 MOVEMENT DISORDERS 1583, 1583 (2014); Teri R. Thomsen & Robert L. Rodnitzky, Juvenile Parkinsonism: Epidemiology, Diagnosis and Treatment, 24(6) CNS DRUGS 467, 469-70 (2010).

⁴³ Thomsen, supra note 42, at 467.

40. The causes of Alzheimer's disease are unknown, and it is believed that Alzheimer's develops as a result of multiple factors rather than a single cause.⁴⁴ Risk factors include: genetic mutations; age; moderate traumatic brain injury (defined as loss of consciousness or amnesia that lasts more than 30 minutes); cardiovascular disease risk factors such as smoking, obesity and diabetes; level of education; and level of social and cognitive engagement.⁴⁵ Alzheimer's disease is also a disease of the elderly. Only approximately 3.8% of Americans diagnosed with Alzheimer's disease are younger than 65 years old, and the vast majority of Americans with Alzheimer's disease -- 81% -- are age 75 or older.⁴⁶

41. Individuals diagnosed with PCS would account for the vast majority of people who fall into Mr. Nichols' class definition, and that number is likely to be very small given that approximately 99% of concussions self-resolve completely. In the unlikely event that a member of Mr. Nichols' proposed class has been diagnosed with early onset of chronic neurocognitive impairment, dementia, Alzheimer's disease, Parkinson's disease, ALS or CTE, he or she almost certainly also would have been diagnosed with PCS.

42. Mr. Nichols has claimed to have brain calcifications that may (or may not) have been caused by brain trauma sustained during his college football career. Brain calcifications are common in the human brain and most often occur spontaneously. Depending on their location, size, and other characteristics, they may be normal.

43. Brain calcifications can be associated with brain infection, vascular disease, tumors, and other conditions, but are not believed to be sequelae for brain trauma or concussions.

⁴⁴ Alzheimer's Ass'n, 2015 Alzheimer's Disease Facts and Figures, 11(3) ALZHEIMER'S & DEMENTIA 332, 336 (2015).

⁴⁵ Id. at 336-37.

⁴⁶ Id. at 340.

44. In general, brain calcifications do not cause symptoms absent some other ailment, such as an infection or a brain tumor.

45. Brain trauma does not cause infections or brain tumors.

I declare under penalty of perjury under the laws of the state of Illinois that the foregoing is true and correct.

EXECUTED on this 14 day of September, 2015, at Chicago, Illinois.

A handwritten signature in black ink, appearing to read "Julian Bailes", written over a horizontal line.

Julian E. Bailes, M.D.

BAILES EXHIBIT 1

CURRICULUM VITAE

JULIAN E. BAILES, JR. M.D.

PERSONAL

Birthplace: Alexandria, Louisiana

Address: Department of Neurosurgery
NorthShore University HealthSystem
2650 Ridge Avenue
Kellogg-3rd Floor
Evanston, IL 60201

EDUCATION

College: Louisiana State University
Baton Rouge, Louisiana
B.S. 1978

Graduate: Louisiana State University School of Medicine
New Orleans, Louisiana
M.D. 1982

Postgraduate: Externship: Neurosurgery – Head/Spinal Trauma
Los Angeles County Hospital
Los Angeles, CA
1981

Externship: Neurosurgical Oncology
Memorial Sloan Kettering Cancer Center
New York, NY
1981

Internship: General Surgery
Northwestern Memorial Hospital
Chicago, Illinois
1982-1983

Residency: Neurological Surgery
Northwestern University Medical Center
Chicago, Illinois
1983-1987

Fellowship: Cerebrovascular Surgery
Barrow Neurological Institute
Phoenix, Arizona
January-July 1988

APPOINTMENTS:

Clinical Instructor
Division of Neurosurgery
Northwestern University
Chicago, Illinois
1987

Chief, Cerebrovascular Surgery
Allegheny General Hospital
Pittsburgh, Pennsylvania
1988 - 1997

Assistant Professor
Division of Neurosurgery
Medical College of Pennsylvania
Philadelphia, Pennsylvania
1989 - 1994

Clinical Instructor
Department of Neurosurgery
West Virginia University
Morgantown, West Virginia
1989 - 1993

Clinical Assistant Professor
Department of Neurosurgery
West Virginia University
1993 - 1997

Associate Professor
Division of Neurosurgery
Medical College of Pennsylvania/Hahnemann Univ.
Philadelphia, Pennsylvania
1994 - 1997

Senior Vice President
Medical Director
Orlando Regional Healthcare System
CareLink Management
Orlando, Florida
1997 - 1998

President/CEO
Greater Orlando Neurosurgery & Spine, P.A.
Orlando, Florida
1998 - 2000

Medical Director
Emergency Medical Services
Osceola County, Florida (Greater Metropolitan Orlando, selected by County Commission)
1998 - 2000

Director, Neurosurgery
Disneyworld Celebration Hospital
Orlando, Florida
1999 - 2000

Assistant Professor, College of Health and Public Affairs
University of Central Florida
Orlando, Florida
1999 - 2000

Professor and Chairman, Department of Neurosurgery
West Virginia University School of Medicine
Morgantown, WV
2000 –2011

Bennett Tarkington Chairman
Department of Neurosurgery
Co-Director, NorthShore Neurological Institute
NorthShore University Health System
Clinical Professor of Neurosurgery
University of Chicago Pritzker School of Medicine
Evanston, IL
2011-present

Co-Chairman, West Virginia Governor's Task Force on Healthcare
2005

Chairman, West Virginia Health Information Network
2006 to 2011

Chairman, Sports Medicine Committee, American Association of Neurological Surgeons & Congress of Neurological Surgeons 2002 to present

Advisor, NFL Players Association Committee on Head Injuries, 2007 to present

NASA Crew Protection Work Group, Orion Mars-Lunar Mission, Houston, TX, 2007-2009

Advisor, Competitive Safeguards & Medical Aspects of Safety Committee, NCAA, 2009 to present

Board of Directors, Blanchette Rockefeller Neurosciences Institute, 2009 to 2011

Medical Director, Spokesman, Progesterone for severe TBI, FDA-approved study, BHR Pharma, 2010-2011.

Chairman, Medical Advisory Board, Pop Warner Football, Inc. Philadelphia, PA. 2010 to present

Director, NFL Players Association, Second Opinion Network. 2010 to present

Neurological Consultant, NCAA

Neurological Consultant, Southeastern Conference

Neurological Consultant, Arena Football League

Board of Directors, Chicago Interurban Neurosurgical Society, 2013 to present

Secretary/Treasurer, American Association of Neurological Surgeons/Congress of Neurological Surgeons Section Neurotrauma and Critical Care, 2014 to present

West Virginia Athletic Commission, 2009-2014

AWARDS

Outstanding Radiology Student, Louisiana State University School of Medicine
New Orleans, Louisiana, 1982.

Anne Addington Research Award, Northwestern University, Chicago, Illinois, 1984.

Health Hero Award for Healthcare Innovations in Pennsylvania, Pittsburgh Business Times, Pittsburgh, 1996. (Telemedicine)

Outstanding Healthcare Achievement of 1996, Pittsburgh Executive Report Magazine 1996.
(Telemedicine)

Finalist, Cerebral Resuscitation Project Competition, National Association of Emergency Medical Physicians, 1996. "Hypothermic Blood Substitutes to Resuscitate from Hemorrhagic Shock."

Dean's Excellence Award for Clinical Service, West Virginia University School of Medicine. 2001, 2003.

Leica Visionary in Neurosurgery Award, 2004.

"America's Best Doctors" 2001-02, 2003-04, 2005-06, 2007-08, 2009-10. 2011, 2012, 2013

"America's Top Surgeons" 2006, 2007, 2009, 2010, 2011, 2012, 2013

Louisiana State University Hall of Distinction Inductee 2011 (highest university honor granted to alumnus)

"Chicago Top Neurosurgeons", Chicago Magazine 2014

"Courage Award" The Association of Boxing Commissions Medical Committee Annual Meeting, Clearwater Beach, FL. July 28, 2014

MEDIA APPEARANCES

ESPN, NBC Sports, Good Morning America, CNN, CNN Sports, Anderson Cooper Show, CBS Morning Show, Outside the Lines, C-Span, Today Show, NBC Nightly News, Larry King Live, PBS, Dr. Oz Show, ABC Nightline, Al Jazeera, ABC Secrets of Your Mind mini-series, ESPN Magazine, Northwest Herald, Time, Sports Illustrated, and various local media.

GRANTS – Principal or Co-Investigator

1986	Study of Effects of CO2 Laser on Primate Peripheral Nerves	Marshall Bennett Fund, Evanston Hospital, Evanston, IL	1986\$30,000
1989	Study of Ultraprofound Hypothermia with blood substitution in canine model	Allegheny-Singer Research Institute, Pittsburgh, PA	\$5,000
1989	Study of effects of hypothermia and blood substitution in multiorgan systems	Cryomedical Sciences, Inc., Washington, D.C.	\$1,000,000
1993	Shock trauma model of hypothermia and blood substitution	Cryomedical Sciences, Inc., Bethesda, MD	\$135,000
1993	Cardiac effects of intracranial hemorrhage	Allegheny-Singer Research Institute, Pittsburgh, PA	\$10,000
1995	National Medical Practice Knowledge Bank, NIST-ATP Grant No. 70NANB5H1183	U.S. Department of Commerce	\$21,300,000
1995	Tirilizad In Subarachnoid Hemorrhage, Principal Investigator	Upjohn, Inc	\$600,000
1996	Telemedicine for Rural Health Care Delivery, Clinical Principal Investigator	U.S. Department of Commerce	\$450,000
1996	Hypothermia and Blood Substitution - Canine and Primate Models, \$100,000	Allegheny-Singer Research Institute	\$100,000
1997	Head Injuries in Professional Football Players, annual to present	NFLPA Players Association	\$135,000
1999	National Center for Study of Concussion in NFL Players	Celebration Health	\$5,000
1999	Teaching Models of Cardiac Cerebral and Trauma Resuscitation	Osceola County Fire-Rescue Service	\$15,000
2003	Medical Models of Homeland Security	Conaway Group	\$25,000
2004	NFL Players' Association Center for Study of Retired NFL Athletes	Medtronic, Inc.	\$250,000
2004	Homeland Security Comprehensive Assessment Model	U.S. Department of Homeland Security	\$700,000
2005	Skull Base Lab Research	Synthes, Inc.	\$30,000
2006	Omega 3 Fatty Acids in Traumatic Brain Injury	Inflammation Research Foundation	\$30,000
2006	Skull Base Lab Research	Synthes, Inc.	\$30,000
2007	Omega 3 Fatty Acids for Traumatic Brain Injury	Martek, Inc.	\$30,000
2007	WVU Neurosurgical Chair	Hazel Ruby McQuain Foundation	\$1,500,000
2007	DHA Supplementation for Prevention of Cognitive Deficits in Retired NFL Players	Martek, Inc.	\$350,000
2007	Skull Base Lab Research	Synthes, Inc	\$30,000
2008	WV State-wide Stroke Network	Hazel Ruby McQuain Foundation	\$300,000
2008	DHA Pre-Treatment for TBI	Martek, Inc.	\$76,000
2008	Skull Base Lab Research	Synthes, Inc	\$30,000
2009	Skull Base Lab Research	Synthes, Inc	\$30,000
2010	Progesterone	BHR Pharma	\$61,000
2013	Concussion Models/Treatment	Abbott Laboratories	\$135,000
2014	TBI Large Animal Model	Q30 Labs	\$66,300

Total Grant Funding

27,458,300

MEMBERSHIPS

American Medical Association
Congress of Neurological Surgeons
American Association of Neurological Surgeons
Pennsylvania Medical Society
Allegheny County Medical Society
Society of Cryobiology
Research Society of Neurological Surgeons
Joint Section AANS/CNS Neurotrauma and Critical Care
Joint Section AANS/CNS Cerebrovascular Surgery
Neurosurgeons for Health Care Reform
Aequanimitas Neurosurgical Society, President
Executive Council, Joint Section AANS/CNS for Trauma & Critical Care
Executive Council, AANS/CNS Joint Section of Cerebrovascular Surgery
West Virginia State Medical Association
American College of Emergency Physicians
Monongalia County Medical Society
West Virginia Emergency Medical Services Council

ADMINISTRATIVE (Institutional and Clinical)

Medical Director, Allegheny General Hospital Telemedicine
Medical Director, Neurolink Telemedicine
Medical Director, Allegheny Physician Access
Chairman, AHERF Committee on Telemedicine
Neurosurgical Consultant, Pittsburgh Steelers Football Team 1988 to 1997
Chairman, Neurosurgical ICU Critical Care Committee 1988 - 1991
Medical Advisory Board, E-Systems Medical Electronics
Neurosurgical Consultant, University of Central Florida, 1998-99
Florida Association of Emergency Medical Services Directors
Medical Director, Carelink Management Nursing Homes and Home Health Care Agency, Orlando, FL 1997-98
Carelink Advisor for Care Utilization, Prescription Drugs, Medicare Regulations. 1997-98.
Medical Director, Center for Study of Head Injuries in Professional Athletes, University of North Carolina, Chapel Hill.
Medical Director, Emergency Medical Services, Ocala County, Florida. 1998-2000
Medical Director, Emergency Medical Services, City of Kissimmee, Florida 1998-2000
Medical Director, Emergency Medical Services, City of St Cloud, Florida 1998-2000
Medical Advisor, Orange Co FL Sheriff's Office and National Sheriffs Association
Medical Director, National Domestic Preparedness Partnership (WVNG, WVSP) 2002-Present
Medical Advisor, Joint Agency Anti-Drug Task Force, Region Five WV 2002-Present

Medical Advisor, West Virginia State Police 2002-Present
Medical Director, Homeland Security Comprehensive Assessment Model, Orange County, FL, 2002-Present.
Chairman, Sports Medicine AANS/CNS Section on Trauma, 2005-present
National Task Force for Transport of Injured Athletes
Examiner, American Board of Neurological Surgeons, 2000, 2004, 2010.
Medical Review Committee, Association of Boxing Commissions, 2010.

NATIONAL / INTERNATIONAL

Program Chairman, AANS/CNS Joint Section of Cerebrovascular Surgery, AANS Meeting Apr, 1996.
Program Director: Sports Related Concussion and Nervous System Injury. Orlando, FL, Feb, 1997.
Program Director: Sports Related Concussion and Nervous System Injury, Orlando, FL, Mar, 1988
Program Director: Sports Related Concussion and Nervous System Injury, Orlando, FL, May 1999
Scientific Committee, Joint Section on Cerebrovascular Surgery, Annual Meeting, Orlando, FL, Feb, 1998
Chairman, Internet Access Committee, Congress of Neurological Surgeons Annual Meeting, New Orleans, LA September, 1997
Cerebrovascular Section Representative to "Neurosurgery On-Call", Internet Access
Chairman, Scientific Advisory Committee, Cryomedical Sciences, Inc.
Chairman, Host Committee, 1991 Congress of Neurological Surgeons
Chairman, Videotape Library, 1992 Congress of Neurological Surgeons
Chairman, Steering Committee on Telemedicine, AHERF
Chairman, Medical Advisory Committee, NMPKB project
Member, State Trauma System Action Team, Charleston, WV, 2002
President, West Virginia Neurosurgical Society, 2002-Present
President, Neurosurgical Society of The Virginias, 2004-2006

EDITORIAL

Editor, Neurosurgery (2009 to present)
Editorial Board, Journal of Neurotrauma
Editorial Board, Computerworld, (1996-1997)
Editorial Board, Telemedicine and e-Health (2002 – present)
Editorial Board, Neurosurgery On-Call (1996-2002)
Editorial Board, CNS/AANS Videotape Library (1989-1993)
Editor, Allegheny General Hospital Neuroscience Journal (1989-1993)
Editorial Board, Journal of Reconstructive Microsurgery Romania (1996-2002)
Editorial Board, AANS/CNS Publications Committee (1996-1999)
Reviewer, Neurosurgery
Reviewer, Journal of Neurotrauma
Reviewer, Stroke
Reviewer, American College of Sports Medicine
Reviewer, Journal of Orthopaedic and Sports Physical Therapy
Reviewer, Sports Medicine (Australia)
Reviewer, Athletic Training

Reviewer, Physician and Sports Medicine
Reviewer, Canadian Medical Journal
Reviewer, Zentralblatt für Neurochirurgie

LICENSURE

State	Year	Cert. No.
Louisiana	1982	016562
Illinois	1984	069762-1
Arizona	1988	17274
Pennsylvania	1988	041673E
Ohio	1996	35-07-1901
Florida	1998	ME0075152
West Virginia	2000	20146

CERTIFICATION

American Board of Neurological Surgery
Certificate No. 92062
Written Passed 1986
Oral Passed 1992

PUBLICATIONS

ABSTRACTS

1. Kwaan HC, Bailes JE, Quigley MR, Cerullo LJ: Analysis of microvascular anastomosis with the low-power CO laser. Fed Proc. #7310, 44:1661, 1985.
2. Quigley MR, Bailes JE, Kwaan HC, Cerullo LJ: Incidence and histology of aneurysms following laser-assisted vascular anastomosis. Thromb Haemostasis #P822, 54:139, 1985.
3. Quigley MR, Bailes JE, Kwaan HC, Cerullo LJ: Endothelial function in laser-assisted microvascular anastomosis. Thromb Haemostasis #P831, 54:141, 1985.
4. Quigley MR, Bailes JE, Kwaan HC, Cerullo LJ: Effect of prostacyclin on surgically-induced vascular trauma. Thromb Haemostasis #P1244, 54:211, 1985.
5. Quigley MR, Bailes JE, Kwaan HC, Cerullo LJ: Laser-assisted vascular anastomosis. Bull Am Phys Soc 30:1854, (RJ9), 1985.
6. Bailes JE, Quigley MR, Cerullo LJ, Kline DG, Sahgal V: Nerve anastomosis with low-power CO2 laser. Bull Am Phys Soc 30:1954 (RJ5), 1985.
7. Quigley MR, Bailes JE, Kwaan HC, Heiferman K, Cerullo LJ: Microvascular laser-assisted anastomosis: Results at one year. Lasers Surg Med #112, 6:179, 1986.

8. Quigley MR, Bailes JE, Molteni A, Brizio-Molteni L, Cerullo LJ: Distant effects of ND-YAD laser. *Lasers Surg Med* #134, 6:206, 1986.
9. Bailes JE, Quigley MR, Cerullo LJ, Kline DG, Shagal V: CO2 laser nerve anastomosis: Histologic and electrophysiologic analysis. *Lasers Surg Med* #157, 6:248. 1986.
10. Quigley MR, Bailes JE, Kwaan HC, Cerullo LJ: Laser-assisted end-to-side anastomosis. *J Reconst Microsurg* 3:75, 1986.
11. Shah A, Bailes JE, Sahgal V, Cerullo LJ: Cytochemistry and morphometry of ratsciatic nerve following laser and suture treatments. *Proceedings American Electron Microscopic Association* 8:56-57, 1986.
12. Bailes JE, Quigley MR, Keenan V, Cerullo LJ, Meyer PR: Head trauma in the spinal-injured patient. *Paraplegia* 25:51 1987.
13. Bailes JE, Spetzler RF, Hadley MN, Baldwin H, Zambramski J: Management morbidity and mortality of Hunt and Hess Grade 4 and 5 aneurysm patients. *J Neurosurg* 70:309A, 1989.
14. Maroon JC, Kennerdell J, Sternau L, Wilberger JE, Bailes JE: The management of recurrent sphenno-orbital meningiomas. *J Neurosurg* 72:354A, 1990.
15. Herman JE, Bailes JE, Quigley MR, Cerullo LJ, Meyer PR Jr: Cervical spine diving injuries. *J Neurosurg* 338-339A, 1990.
16. Teeple E, Bailes JE, Leavitt ML, Shih SR, Marquardt M, Elrifai A, Maroon JC: Ultra-profound hypothermia, complete blood substitution, cardiac arrest and low-flow perfusion in a dog model: A report. *J Neurosurg Anesth* 2:515, 1990.
17. Leavitt ML, Bailes JE, Elrifai AM, Teeple E, Shih RS, Marquardt M, Maroon JC: Survival from prolonged cardiac arrest in totally exsanguinated hypothermic dogs. *Fed Amer Soc Exp Biol J* 4(4):A963, 1990.
18. Bailes JE, Leavitt ML, Maroon JC, Teeple E, Elrifai AM, Shih SR, Marquardt M: Potential for ultra-profound hypothermia in totally exsanguinated and blood-substituted canine model: I. The method. *Cryobiology* 27(6):622, 1990.
19. Elrifai AM, Bailes JE, Leavitt ML, Shih RS, Teeple E, Marquardt M, Maroon JC: The use of ultra-profound hypothermia in totally exsanguinated and blood substituted canine model: II. The outcome. *Cryobiology* 27(6):622-623, 1990.
20. Elrifai AM, Bailes JE, Leavitt ML, Shih RS, Teeple E, Marquardt M, Maroon JC: The use of blood substitutes for whole body perfusion in ultra-profound hypothermic cardiac arrest. *An Clin Lab Med Sci* 20(4):292, 1990.
21. Elrifai AM, Loesch DV, Leavitt ML, Bailes JE, Shih SR, Maroon JC, Ciongoli KA, Devenyi C: rewarming rate and survival post induced ultra-profound hypothermia. *Clin Research* 38(3):793A, 1990.
22. Leavitt ML, Bailes JE, Shih RS, Elrifai AM, Teeple E, Maroon JC: Outcome following cooling to near the freezing point in totally blood-substituted dogs. *Soc Neuroscience Abst* 16:740, 1990.

23. Shih SR, Gianaris PG, Bailes JE, Maroon JC: CO2 laser-assisted embryonic spinal cord transplantation I. *Lasers Surg Med* 53:31, 1991.
24. Gianaris PG, Shih SR, Bailes JE, Maroon JC: CO2 laser-assisted embryonic spinal cord transplantation II. *Lasers Surg Med* 53:31-32, 1991.
25. Bailes JE, Spetzler RF: Management of symptomatic internal carotid artery occlusion. *J Neurosurg* 24:352-353A, 1991.
26. Leavitt ML, Bailes JE, Elrifai AM, Shih TS, Devenyi C, Ciongoli K, Bazmi B, Maroon JC: Experimental total blood substitution during profound hypothermic cardiac arrest in dogs. *Cryobiology* 28(6):520, 1991.
27. Leavitt ML, Bailes JE, Shih RS, Elrifai AM, Ciongoli K, Devenyi C, Bazmi B, Maroon JC: Complete blood substitution during profound hypothermic cardiac arrest in dogs. *Biomater Art Cells & Immob Biotech* 19:415, 1991.
28. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Taylor MJ: Brain temperature in profound hypothermia: An experimental observation. *Clin Res* 39:798A, 1991.
29. Elrifai AM, Bailes JE, Leavitt ML, Shih SR, Teeple E, Taylor MJ, Maroon JC: In vivo changes in the composition of a blood substitution of a blood substitute during hypothermia. *Proceedings of Assoc Adv Med Instrument* 169, 1991.
30. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Devenyi C, Ciongoli K, Bazmi B: Continuous measurements of intracranial pressure under hypothermia and blood substitution in canines. *Clin Res* 40(1):107A, 1992.
31. Bailes JE, Elrifai AM, Taylor MJ, Leavitt ML, Shih SR, Teeple E, Maroon JC: Blood substitution in profound hypothermia. *ASAIO Abstr*, p 4, 1992.
32. Leavitt ML, Bailes JE, Teeple E, Taylor MJ, Elrifai AM, Shih SR, Maroon JC, Ciongoli K, Devenyi C, Bazmi B: Improved method for profound hypothermic cardiac arrest using blood substitution. *ASAIO Abstr*, p 58, 1992.
33. Leavitt ML, Bailes JE, Elrifai AM, Taylor MJ, Maroon JC, Shih TS, Teeple E: Blood parameters following extracorporeal circulation of a blood substitute during profound hypothermia in dogs. *American Academy of Cardiovascular Perfusion Abstr*, 1992.
34. Leavitt ML, Bailes JE, Shih RS, Elrifai AM, Teeple E, Taylor MJ, Maroon JC: Changes in intracranial pressure during profound hypothermic blood substitution in dogs. *Fed Amer Soc Exp Biol J*, 6(5):A831, 1992.
35. Teeple E, Elrifai AM, Bailes JE, Maroon JC, Leavitt ML, Shih SR, Bazmi B: Brain versus body temperature in hypothermia: Clinical implications derived from an experimental study. *Soc Cardiovasc Anesthesiologists Proceedings*, p 274, 1992.
36. Bailes JE, Elrifai AM, Leavitt ML, Shih SR, Teeple E, Taylor MJ, Maroon JC: Blood substitution and profound hypothermia: Extending the safe limits of cardiac arrest. *Aviat Space Environ Med* 63(5):408, 1992.
37. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Ciongoli K, Devenyi C, Bazmi B: The effects of rewarming on influencing variable outcomes after exposure to ultraprofound

hypothermia. Proceedings Assoc Adv Med Instrum p 26, 1992.

38. Leavitt ML, Bailes JE, Shih SR, Elrifai AM, Taylor MJ, Maroon JC, Teeple E: Rewarming following profound hypothermia combined with a sanguineous extracorporeal circulation in canines. Les Journees du CECEC Programme, Communication N 201, 1992.
39. Leavitt ML, Bailes JE, Shih SR, Taylor MJ, Elrifai AM, Teeple E, Maroon JC, Devenyi C, Ciongoli K, Bazmi B: Temperature changes after profound hypothermic cardiac arrest. World Congress of Anesthesiologists Abstracts, p. 126, 1992.
40. Leavitt ML, Bailes JE, Shih SR, Taylor MJ, Elrifai AM, Teeple E, Maroon JC, Devenyi C, Ciongoli K, Bazmi B: Brain versus body temperature changes after profound hypothermic cardiac arrest. Soc Neuroscience Abstr 18(2):1586, 1992.
41. Elrifai AM, Bailes JE, Teeple E, Leavitt ML, Shih SR, Taylor MJ, Maroon JC: Serum levels of creatinine kinase (CK) in hypothermia and complete blood substitution. Soc Neuroscience Abstr 18(2):1586, 1992.
42. Teeple E, Elrifai AM, Bailes JE, Shih SR, Leavitt ML, Taylor MJ, Maroon JC, Ciongoli KA, Devenyi C, Bazmi B: A summary of the means of support required during resuscitation from profound hypothermia and complete blood substitution. Proceed Int Soc Appl Cardiovasc Biology, Group A, P#10, 1992.
43. Leavitt ML, Bailes JE, Taylor MJ, Elrifai AM, Shih SR, Teeple E, Maroon JC: Low flow extracorporeal circulation in totally blood substituted profoundly hypothermic dogs. Proceed Int Soc Appl Cardiovasc Biology, Group A, P#6, 1992.
44. Elrifai AM, Bailes JE, Teeple E, Shih SR, Taylor MJ, Leavitt ML, Maroon JC: Cerebral perfusion pressure in profound hypothermic cardiac arrest in canines. Anesth Analg 76(2):S381, 1993.
45. Leavitt ML, Bailes JE, Elrifai AM, Taylor MJ, Teeple E, Shih RS, Maroon JC: Asanguineous perfusion of profoundly hypothermic dogs: An update. American Academy of Cardiovascular Perfusion Abstracts, 1993.
46. Leavitt ML, Bailes JE, Taylor MJ, Elrifai AM, Teeple E, Shih SR, Maroon JC: Biochemical recovery following profound hypothermic cardiac arrest and blood substitution. ASAIO Abstr. 22:64, 1993.
47. Leavitt ML, Bailes JE, Taylor MJ, Shih RS, Elrifai AM, Teeple E, Maroon JC: Motor activity during re-warming subsequent to profound hypothermic cardiac arrest in totally blood substituted dogs. Fed Amer Soc Exp Biol J 7(3):A441, 1993.
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49. Bailes JE, Elrifai AM, Teeple E, Taylor MJ, Shih SR, Leavitt ML, Maroon JC: The value of monitoring brain temperature, intracranial pressure and cerebral perfusion pressure during hypothermia and cardiopulmonary bypass with cardiac arrest. Eur J Biomed Technol. 15(4):202, 1993.
50. Taylor MJ, Elrifai AM, Bailes JE, Shih SR, Teeple E, Leavitt ML, Maroon JC, Ciongoli KA, Devenyi C: The use of aqueous blood substitution during 3 hours of experimental hypothermic

cardiac arrest. Eur J Biomed Technol. 15(4):205, 1993.

51. Taylor MJ, Bailes JE, Elrifai AM, Shih SR, Teeple E, Leavitt ML, Baust JG, Maroon JC: New aqueous blood substitutes for general tissue preservation during 3 hours of profound hypothermic cardiac arrest: I. Solution design and biochemical outcome. Cryobiology 30(6):655-656, 1993.
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54. Elrifai AM, Bailes JE, Teeple E, Taylor MJ, Shih SR, Leavitt ML, Maroon JC: Monitoring cerebral perfusion pressure in experimental profound hypothermia and cardiac arrest. Cryobiology 30(6):643, 1993.
55. Elrifai AM, Bailes JE, Govindan S, Teeple E, Taylor MJ, Shih SR, Leavitt ML, Adatepe MH, Maroon JC: Evaluation of cerebral blood flow using radioactive Xenon133 after long term survival of animals exposed to profound hypothermia and complete blood substitution. Soc Neuroscience Abst 19(2):1221, 1993.
56. Taylor MJ, Bailes JE, Elrifai AM, Shih SR, Teeple E, Leavitt ML, Baust JG, Maroon JC: Cellular protection using new hypothermic blood substitutes during 3 hr cardiac arrest. ASAIO Abst, p 58, 1994.
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58. Elrifai AM, Bailes JE, Govindan S, Leavitt ML, Teeple E, Taylor MJ, Shih SR, Adatepe MH, Maroon JC: Cerebral blood flow measurement using radioactive Xenon 133 under Pentothal and Fentanyl Anesthesia. Soc Neuroscience Abst, 20(2):1421, 1994.
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64. Bailes JE: Intraoperative microvascular doppler sonography in aneurysm surgery. *Stroke* 28:1854, 1997.
65. Elrifai A, Taylor MJ, Bailes JE, Shih Sr: Further development of ultraprofound hypothermia and blood substitution in a canine model with a view to clinical trials. *Cryobiology* 1998.
66. Bailes JE: The neurosurgeon's future role in emergency medical services. *Neurosurgery* 45: 708-709, 1999.
67. Bailes JE: Experience with Transient Spinal Cord injury in athletes. *Neurosurgery* 55:486-487. 2004.
68. Bailes JE, Mills J, Wilson J, et al: Omega 3 fatty acid supplementation reduces the extent of axon damage after brain trauma. ISSFAL 2008 website.
69. Bailes JE, Patel V: The potential for DHA to mitigate mild traumatic brain injury. *Military Medicine* 179(11 Suppl) 112-6, DOI:10.7205/MilMed-D-14-00139, 2014
70. Elias J, Bailes J. Early experience with trans-sulcal parafascicular Exoscopic resection of supratentorial brain tumors. *Neuro Oncol* (2014) 16 (suppl 5):v161. doi: 10.1093/neuonc/nou265.13.
71. Bailes JE, Elias J, Merrell R, O'Leary S, Stereotactic Laser Thermal Ablation of Recurrent Posterior Fossa Metastatic Lesion: Description of New Technology for Infratentorial tumors refractory to conventional therapies. *Neuro Oncol* (2014) 16 (suppl 5): v162. doi: 10.1093/neuonc/nou265.14

EDITORIAL COMMENTS

1. Bailes JE: Comment on Frim et al - Thromboembolism prophylaxis in neurosurgery. *Neurosurgery* 30:832-833, 1992.
2. Bailes JE: Comment on Cantu RC, Mueller FO - Catastrophic football injuries: 1977-1998. *Neurosurgery* 47:673-677, 2000.
3. Bailes JE: Comment on Collins MW, Lowell MR, Iverson GL, et al. Cumulative effects of concussion in high school athletes. *Neurosurgery*, in press, 2002.
4. Bailes JE: Comment on McCrea M et al: Immediate neurocognitive effects of concussion. *Neurosurgery* 50:1041, 2002.
5. Bailes JE, Cantu RC, Day AL: Reply to comments by Kreider et al, on the editorial: The Neurosurgeon in Sport: Awareness of the Risks of Heatstroke and Dietary Supplements. *Neurosurgery* 52: 255-257. 2003.
6. Bailes JE: Comment on Collins MW, Lovell MR, Everson GL, et al: Cumulative Effects of Concussion in Athletes. *Neurosurgery* 51: 1180. 2002
7. Bailes JE: Reply to Tsuzuki et al, on Prabhu VC, Bailes JE article: Chronic subdural hematoma

complicating arachnoid cyst secondary to soccer-related head injury: Case report. *Neurosurgery* 53:243. 2003.

8. Bailes JE: Comment on Pellman et al: Concussion in professional football: epidemiological features of game injuries and review of the literature – part 3. *Neurosurgery* 54:81-96. 2004.
9. Bailes JE: Comment on Bleiberg et al: Duration of cognitive impairment after sports concussion. *Neurosurgery* 54:1073-1080. 2004.
10. Bailes JE: Comment on Blount et al: Sports and pediatric cerebrospinal fluid shunts: who can play? *Neurosurgery* 54:1190-1198. 2004.
11. Miele VJ, Bailes JE: Comment on Levy et al: Analysis and evolution of head injury in football. *Neurosurgery* 55: 649-655. 2004
12. Bailes JE: Comment on Levy et al: Birth and evolution of the football helmet. *Neurosurgery* 55: 656-662. 2004.
13. Bailes JE: Comment on Pellman et al: Concussion in professional football: repeat injuries – part 4. *Neurosurgery* 55:860-876. 2004
14. Bailes JE: Comment on Hoshizaki TB, Brien SE: The science and design of head protection in sport. *Neurosurgery* 55:956-967. 2004
15. Bailes JE: Comment on Pellman et al: Concussion in professional football involving 7 or more days out – part 5. *Neurosurgery* 55:1100-1119. 2004
16. Bailes JE: Comment on Pellman et al: Concussion in professional football: players returning to the same game – Part 7. *Neurosurgery* 56: 90. 2005.
17. Bailes JE, Miele JE: Fatal attraction for the ring (invited opinion editorial). *New York Times*, May 22, 2005 Sec. 8 p. 9.
18. Bailes JE: Comment on Moser et al: Prolonged effects of concussion in high school athletes. *Neurosurgery* 57:306. 2005.
19. Bailes JE: Comment on Viano et al: Concussion in professional football: brain responses by finite element analysis: part 9. *Neurosurgery* 57:916. 2005.
20. Bailes JE: Comment on Gosselin et al: Neurophysiological anomalies in symptomatic and asymptomatic concussed athletes. *Neurosurgery* 58: 1160. 2006.
21. Bailes JE: Comment on Viano et al: Concussion in professional football: performance of newer helmets in reconstructed game impacts – part 13. *Neurosurgery* 59:606. 2006
22. Bailes JE: Comment on Omalu et al: Chronic traumatic encephalopathy in a National Football League player: part II. *Neurosurgery* 59:1093. 2006.
23. Bailes JE: Comment on Viano et al: Concussion in professional football: biomechanics of the struck player – part 14. *Neurosurgery* 61: 327-328. 2007.
24. Bailes JE: Comment on Slobounov et al: Differential rate of recovery in athletes after first and second concussion episodes. *Neurosurgery* 61: 344. 2007.

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96. Bailes JE: Management of poor-grade aneurysm patients. Defusing the Bomb, Brain Aneurysms: The New Era. Harvard University, Boston, MA, June 1991.
97. Bailes JE: Carotid endarterectomy. Medical Grand Rounds, Latrobe Hospital, Latrobe, PA, June 1991.
98. Leavitt ML, Bailes JE, Elrifai AM, Shih SR, Devenyi C, Ciongoli K, Bazmi B, Maroon JC: Experimental total blood substitution during profound hypothermic cardiac arrest in dogs. International Society Cryobiology, July 1991.
99. Leavitt ML, Bailes JE, Shih RS, Elrifai AM, Ciongoli K, Devenyi C, Bazmi B, Maroon JC: Complete blood substitution during profound hypothermic cardiac arrest in dogs. IV International Symposium on Blood Substitutes, August 1991.
100. Bailes JE: New concepts in the treatment of stroke. Tenth annual internal medicine conference. Cambria-Somerset Council, Seven Springs, PA, September 1991.
101. Bailes JE: Cardiac effects of intracranial hemorrhage. Congress Neurological Surgeons, Orlando, FL, October 1991.
102. Bailes JE, Maroon JC: Transient spinal cord injury in athletes. Congress Neurological

Surgeons, Orlando, FL, October 1991.

103. Bailes JE, Elrifai AM, Maroon JC, Leavitt ML, Shih SR: Neurological outcome in relation to rewarming rates in hypothermic procedures. Congress of Neurological Surgeons, October 1991.
104. Bailes JE: Surgical treatment of stroke. Medical Grand Rounds, Monongahela Valley Hospital, Monongahela, PA, November 1991.
105. Bailes JE: Treatment of stroke. Medical Education Program, Greater Pittsburgh Rehab Hospital, Monroeville, PA, November 1991.
106. Bailes JE: Cardiac effects of intracranial hemorrhage. Neuroscience Conference, Allegheny General Hospital, November 1991.
107. Elrifai AM, Bailes JE, Leavitt ML, Shih SR, Teeple E, Taylor MJ, Maroon JC: In vivo changes in the composition of a blood substitute during hypothermia. Cardiovascular Science & Technology, Bethesda, MD, December 1991.
108. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Taylor MJ: Brain temperature in profound hypothermia: An experimental observation. Southern Meet Am Fed Clin Res, New Orleans, LA, January 1992.
109. Leavitt ML, Bailes JE, Elrifai AM, Taylor MJ, Maroon JC, Shih RS, Teeple E: Blood parameters following extracorporeal circulation of a blood substitute during profound hypothermia in dogs. American Academy of Cardiovascular Perfusion, Orlando, FL, January 1992.
110. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Devenyi C, Ciongoli K, Bazmi B: Continuous measurements of intracranial pressure under hypothermia and blood substitution in canines. Joint Meet. Western Assoc. of Physicians and Am Fed Clin Res, Carmel, CA, February, 1992.
111. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Devenyi C, Ciongoli K, Bazmi B: Rewarming, hypothermia, and cardiopulmonary bypass. 30th Annual International Conference of the American Society for Extra-Corporeal Technology, Washington, DC, March 1992.
112. Elrifai AM, Bailes JE, Shih SR, Leavitt ML, Teeple E, Taylor MJ, Maroon JC: Hemodilution or blood substitution: An experimental study. 30th Annual International Conference of the American Society for Extra-Corporeal Technology, Washington, DC, March 1992.
113. Leavitt ML, Bailes JE, Shih RS, Elrifai AM, Teeple E, Taylor MJ, Maroon JC: Changes in intracranial pressure during profound hypothermic blood substitution in dogs. An Meet Fed Amer Soc Exp Biol, Anaheim, CA, April 1992.
114. Bailes JE: Cardiac effects of intracranial hemorrhage. Amer Assoc Neurol Surg, San Francisco, CA, April 1992.
115. Bailes JE, Elrifai AM, Leavitt ML, Shih SR, Teeple E, Taylor MJ, Maroon JC: Blood substitution and profound hypothermia: Extending the safe limits of cardiac arrest. Annual Meeting of the Aerospace Medical Association, Miami, May 1992.
116. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Taylor MJ: Brain temperature

- in profound hypothermia: An experimental observation. Southern Meeting of American Federation of Clinical Research, May 1992.
117. Leavitt ML, Bailes JE, Taylor MJ, Elrifai AM, Shih SR, Teeple E, Maroon JC: Improved method for profound hypothermic cardiac arrest using blood substitution, American Society for Artificial Internal Organ An meet, Nashville, TN, May 1992.
118. Teeple E, Elrifai AM, Bailes JE, Leavitt ML, Shih SR, Maroon JC, Bazmi B: Brain versus body temperature in hypothermia: Implications derived from an experimental study. An Meet Soc Cardiovasc Anesthesiologists, Boston, MA, May 1992.
119. Elrifai AM, Bailes JE, Govindan S, Leavitt ML, Teeple E, Shih SR, Adatepe MH, Maroon JC: Adaptation of the 10a Cerebrograph for an experimental model. Advances in Cerebral Blood Flow Conference, Pittsburgh, PA, May 1992.
120. Bailes JE: Overview of CBF research and moderation. International Cerebral Blood Flow Conference, Allegheny General Hospital, Pittsburgh, PA, May 1992.
121. Elrifai M, Bailes JE, Maroon JC, Shih SR, Leavitt ML, Teeple E, Devenyi C, Ciongoli K, Bazmi B: The effects of rewarming on influencing variable outcomes after exposure to ultraprofound hypothermia. An Meet Assoc Adv Med Instrum, Anaheim, CA, June 1992.
122. Leavitt ML, Bailes JE, Shih SR, Elrifai AM, Taylor MJ, Maroon JC, Teeple E: Rewarming following profound hypothermia combined with asanguineous extra-corporeal circulation in canines. XVI Ilemes Journees du Cercle D'Etude de la Circulation Extra-Corporelle, Comite Regional Sub-Mediterranee, Paris, France, June 1992.
123. Bailes JE: Surgical treatment of stroke. Grand Rounds, Hamot Medical Center, Erie, PA, June 1992.
124. Bailes JE: Athletic cervical spine injuries. National Athletic Trainers Assoc. Ann. Meeting, Denver, CO, June 1992.
125. Leavitt ML, Bailes JE, Shih SR, Taylor MJ, Elrifai AM, Teeple E, Maroon JC, Devenyi C, Ciongoli K, Bazmi B: Temperature changes after profound hypothermic cardiac arrest. 10th World Congress of Anesthesiologists. The Hague, The Netherlands, June 1992.
126. Bailes JE: Surgical treatment of stroke. Grand Rounds, Elk County Hospital, Ridgeway, PA, September 1992.
127. Bailes JE, Surgical treatment of stroke. Grand Rounds, Clearfield Hospital, Clearfield, PA, September 1992.
128. Bailes JE: Surgical treatment of stroke. Grand Rounds, Somerset Hospital, Somerset, PA, October 1992.
129. Leavitt ML, Bailes JE, Shih SR, Taylor MJ, Elrifai AM, Teeple E, Maroon JC, Devenyi C, Ciongoli K, Bazmi B: Brain versus body temperature changes after profound hypothermic cardiac arrest. Soc Neuroscience, Anaheim, CA, October 1992.
130. Elrifai AM, Bailes JE, Teeple E, Leavitt ML, Shih SR, Taylor MJ, Maroon JC: Serum levels of creatinine kinase (CK) in hypothermia and complete blood substitution. Soc Neuroscience, Anaheim, CA, October 1992.

131. Bailes JE: Vascular relationships of the terminal basilar artery. AGH Neuroscience Journal, Fall 1992.
132. Bailes JE, Vidovich D: Comprehensive approach for repairing basilar artery apex aneurysms. AGH Neuroscience Journal, Fall 1992.
133. Teeple E, Elrifai AM, Bailes JE, Shih SR, Leavitt ML, Taylor MJ, Maroon JC, Ciongoli KA, Devenyi C, Bazmi B: A summary of the means of support required during resuscitation from profound hypothermia and complete blood substitution. Int Soc Appl Cardiovasc Biology, St. Louis, MO, November 1992.
134. Leavitt ML, Bailes JE, Shih SR, Taylor MJ, Elrifai AM, Teeple E, Maroon JC: Low flow extracorporeal circulation in totally blood substituted profoundly hypothermic dogs. Int Soc Appl Cardiovasc Biology, St. Louis, MO, November 1992.
135. Leavitt ML, Bailes JE, Elrifai AM, Taylor MJ, Teeple E, Shih SR, Maroon JC: Asanguineous perfusion of profoundly hypothermic dogs: An update. An Meet American Academy of Cardiovascular Perfusion, San Antonio, TX, January 1993.
136. Bailes JE: Cerebral aneurysms. Dubois Regional Medical Center Staff Conference, Dubois, PA, January 1992.
137. Bailes JE: surgical treatment of stroke. Medical Staff Conf. Armstrong Memorial Hospital, Kittanning, PA, March 1993.
138. Leavitt ML, Bailes JE, Taylor MJ, Shih RS, Elrifai Am, Teeple E, Maroon JC: Motor activity during rewarming subsequent to profound hypothermic cardiac arrest in totally blood substituted dogs. An Meet Fed Amer Soc Exp Biol. New Orleans, LA, March 1993.
139. Elrifai AM, Bailes JE, Teeple E, Shih SR, Taylor MJ, Leavitt ML, Maroon JC: Cerebral perfusion pressure in profound hypothermic cardiac arrest in canines. Int Anesth Res Soc, San Diego, CA, March 1993.
140. Bailes JE: Surgical treatment of stroke. Medical Staff Meeting, Sharon General Hospital, Sharon, PA, April 1993.
141. Bailes JE: Head injuries in athletes. American Assoc. Neurological Surgeons, Boston, MA, April 1993.
142. Maroon JC, Bailes JE, Quigley MR, Wilberger JE, Onik G: Cryosurgical application in the treatment of brain, spinal and orbital tumors. American Assoc Neurological Surgeons, Boston, MA, April 1993.
143. Bailes JE: Cervical spine trauma in athletes. Pennsylvania Athletic Trainers' Society. Hershey, PA, May 1993.
144. Bailes JE: Recognizing and managing cranial trauma in athletes. Pennsylvania Medical Trainers' Society. Hershey, PA, May 1993.
145. Elrifai AM, Bailes JE, Govindan S, Leavitt ML, Teeple E, Shih SR, Taylor MJ, Adatepe MH, Maroon JC: Evaluation of cerebral blood flow using radioactive Xenon133 pre vs post-hypothermia and blood substitution. Int Meet Cereb Blood Flow Metabol. Sendai, Japan, May

1993.

146. Bailes JE: Intraoperative angiography and temporary balloon occlusion of the basilar artery. International Congress on Minimally Invasive Techniques in Neurosurgery. Weisbaden, Germany, June 1993.
147. Taylor MJ, Elrifai AM, Bailes JE, Shih SR, Teeple E, Leavitt ML, Maroon JC, Ciongoli KA, Devenyi C: The use of aqueous blood substitution during 3 hours of experimental hypothermic cardiac arrest. Fifth Eur Cong Extra-Corporeal Technol, Arles, France, June 1993.
148. Taylor MJ, Bailes JE, Elrifai AM, Shih SR, Teeple E, Leavitt ML, Baust JG, Maroon JC: New aqueous blood substitutes for general tissue preservation during 3 hours of profound hypothermic cardiac arrest: I. Solution design and biochemical outcome. CRYO 93, An Meet Cryobiology Soc, Atlanta, GA, July 1993.
149. Elrifai AM, Bailes JE, Taylor MJ, Teeple E, Shih SR, Leavitt ML, Baust JG, Maroon JC: New aqueous blood substitutes for general tissue preservation during 3 hours of profound hypothermic cardiac arrest: I. Methods and Neurological Outcome. CRYO 93, An Meet Cryobiology Soc, Atlanta, GA, July 1993.
150. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Taylor MJ, Teeple E, Leavitt ML, Ciongoli KA: The correlation of rewarming with outcome in an experimental canine model of profound hypothermia involving blood substitution and cardiac arrest. CRYO 93, An Meet Cryobiology Soc, Atlanta, GA, July 1993.
151. Elrifai AM, Bailes JE, Teeple E, Taylor MJ, Shih SR, Leavitt ML, Maroon JC: Monitoring cerebral perfusion pressure in experimental profound hypothermia and cardiac arrest. CRYO 93, An Meet Cryobiology Soc, Atlanta, GA, July 1993.
152. Bailes JE: Perioperative management of subarachnoid hemorrhage. Congress of Neurological Surgeons, Vancouver, BC, October 1993.
153. Bailes JE, Elrifai AM, Taylor MJ, Shih SR, Teeple E, Leavitt ML, Baust JG, Maroon JC: Ultra-profound hypothermia combined with blood substitution: A new protocol for extending the safe limits of cardiac arrest up to 3 hours. Am Coll Surgeon, Surg Forum An Meet, San Francisco, CA, October 1993.
154. Elrifai AM, Bailes JE, Govindan S, Teeple E, Taylor MJ, Shih SR, Leavitt ML, Adatepe MH, Maroon JC: Evaluation of cerebral blood flow using radioactive Xenon133 after long term survival of animals exposed to profound hypothermia and complete blood substitution. An Meet Soc Neuroscience, Washington, DC, November 1993.
155. Bailes JE: Characterization of cardiac rhythm and contractility abnormalities following subarachnoid hemorrhage in canine model. Aequanimitas - Kasdon Winter Meeting, Jackson Hole, WY, March 1994.
156. Bailes JE: Management of athletic head and spinal injuries. Neurological Injuries in Sports Medicine Conference, Orlando, FL, March 1994.
157. Bailes JE: Management of head and spinal "spear" injuries in athletes. American Association of Neurological Surgeons, San Diego, CA, April 1994.
158. Bailes JE, Elrifai AM, Shih SR, Maroon JC, Nellis K: Characterization of the cardiac rhythmic

- and myocardial disturbances that occur following experimentally induced subarachnoid hemorrhage. American Association of Neurological Surgeons, San Diego, CA, April 1994.
159. Bailes JE: Skull base approaches to intracranial aneurysms. ANI Mini Symposium in Skull Base Surgery, Pittsburgh PA, April 1994.
160. Taylor MJ, Bailes JE, Elrifai AM, Shih SR, Teeple E, Leavitt ML, Baust JG, Maroon JC: Cellular protection using new hypothermic blood substitutes during 3 hr cardiac arrest. American Society for Artificial Internal Organ Ann Meet, San Francisco, CA, April 1994.
161. Elrifai AM, Bailes JE, Teeple E, Taylor MJ, Shih SR, Leavitt ML, Maroon JC: Monitoring cerebral perfusion pressure in experimental profound hypothermia and cardiac arrest. Advances in Clinical Aspects of Cerebral Blood Flow Conference, Pittsburgh, PA, April 1994.
162. Elrifai AM, Bailes JE, Gobindan S, Teeple E, Taylor MJ, Shih SR, Leavitt ML, Adatepe MH, Maroon JC: Evaluation of cerebral blood flow using radioactive Xenon133 after long term survival of animals exposed to profound hypothermia and complete blood substitution. Advances in Clinical Aspects of Cerebral Blood Flow Conference, Pittsburgh, PA, April 1994.
163. Elrifai AM, Bailes JE, Diamond DL, Taylor MJ, Simon D, Davis D, Shih SR, Clark RE, Maroon JC: Hemorrhagic shock model of profound hypothermia and complete blood substitution: Transcranial carotid doppler evaluation of cerebral blood flow velocity. Advances in Clinical Aspects of Cerebral Blood Flow Conference, Pittsburgh, PA, April 1994.
164. Taylor MJ, Bailes JE, Elrifai AM, Shih SR, Baust JG, Maroon JC: New approaches to bloodless surgery using complete blood substitution and profound hypothermia. Invited paper at International Resuscitation Research Conference, University of Pittsburgh, Pittsburgh, PA, May 1994.
165. Bailes JE: Minor head injury in athletes. National Athletic Trainers Assoc. Symposium, Washington, DC, June 1994.
166. Bailes JE, Fukushima T: Surgical strategies for paraclinoid giant aneurysms. 6th Annual Meeting Japanese Skull Base Society, Nagoya, Japan. June 1994.
167. Bailes JE: Present and future applications of profound hypothermia in skull base surgery. 6th Annual Meeting Japanese Skull Base Society, Nagoya, Japan. June 1994.
168. Taylor MJ, Simon D, Elrifai AM, Shih SR, Bailes JE, Maroon JC, Diamond DL: A feasibility study in a canine model for using profound hypothermia and blood substitution with Hypothermosol to enable resuscitation after hemorrhagic shock. Ann Meet Soc of Cryobiology, Japan, August 1994.
169. Bailes JE: Treatment options for the acute stroke patient. Grand Rounds, Somerset Hospital, Somerset, PA, August 1994.
170. Taylor MJ, Bailes JE, Elrifai AM: Hypothermia and blood substitution. Research Institute for Brain and Blood Vessels, Akita, Japan, August 1994.
171. Bailes JE: Treatment options for the acute stroke patient: Grand Rounds, Elk County Hospital, Ridgeway, PA, September 1994.
172. Bailes JE: Medical triage using a new telemedicine system: NeuroLink. Charles Cole Memorial

Hospital, Coudersport, PA, September 1994.

173. Bailes JE: NeuroLink: A computerized neurosurgical telemedicine network. Sharon Regional Medical Center Grand Rounds, Sharon, PA, September 1994.
174. Bailes JE: Surgical treatment of cerebral aneurysms and care of the subarachnoid hemorrhage patient. Medical Grand Rounds, Shadyside Hospital, Pittsburgh, PA, September 1994.
175. Elrifai AM, Bailes JE, Teeple E, Taylor MJ, Shih SR, Maroon JC: Monitoring cerebral perfusion pressure in experimental profound hypothermia and cardiac arrest. International Symposium on Hypothermic Medicine, Pittsburgh, PA, September 1994.
176. Elrifai AM, Bailes JE, Diamond DL, Taylor MJ, Simon D, Davis D, Shih SR, Clark RE, Maroon JC: Hemorrhagic shock model of profound hypothermia and complete blood substitution: Transcranial carotid doppler evaluation of cerebral blood flow velocity. International Symposium on Hypothermic Medicine, Pittsburgh, PA, September 1994.
177. Elrifai AM, Bailes JE, Maroon JC, Shih SR, Taylor MJ, Teeple E: The correlation of rewarming with outcome in an experimental canine model of profound hypothermia involving blood substitution and cardiac arrest. International Symposium on Hypothermic Medicine, Pittsburgh, PA, September 1994.
178. Elrifai AM, Bailes JE, Maroon JC, Shih Sr, Teeple E, Taylor MJ: Brain temperature in profound hypothermia: An experimental observation. International Symposium on Hypothermic Medicine, Pittsburgh, PA, September 1994.
179. Taylor MJ, Elrifai AM, Shih SR, Bailes JE, Maroon JC: A feasibility study in a canine model for using profound hypothermia and blood substitution with Hypothermosol to enable resuscitation after hemorrhagic shock. International Symposium on Hypothermic Medicine, Pittsburgh, PA, September 1994.
180. Elrifai AM, Bailes JE, Teeple E, Shih SR, Taylor MJ, Maroon JC: Serum levels of creatinine kinase (CK) in hypothermia and complete blood substitution. International Symposium on Hypothermic Medicine, Pittsburgh, PA, September 1994.
181. Bailes JE: Surgical approaches to basilar trunk and vertebrobasilar aneurysms. Annual Meeting Congress of Neurological Surgeons. Chicago, IL, October 1994.
182. Bailes JE, Maroon JC: Preliminary experience and cost analysis with NeuroLink: A neurosurgical wide area computer network. Annual Meeting Congress of Neurological Surgeons, Chicago, IL, October 1994.
183. Bailes, JE, Elrifai AM, Taylor MJ, Maroon JC, Shih SR: Ultraprofound hypothermia with blood substitution in a shock trauma model. Annual Meeting Congress of Neurological Surgeons, Chicago, IL, October 1994.
184. Elrifai AM, Bailes JE, Govindan S, Teeple E, Taylor MJ, Shih SR, Adatepe MH, Maroon JC: Cerebral blood flow measurement using radioactive Xenon 133 under Pentothal and Fentanyl Anesthesia. Ann Meet Soc Neuroscience, Miami, FL, November 1994.
185. Bailes JE: Surgical approaches to vertebrobasilar junction and basilar trunk aneurysms. ANI Annual Cerebrovascular Symposium, Pittsburgh, PA, December 1994.

186. Bailes JE: Skull base approaches to cerebral aneurysms. Pan-Pacific Skull Base Surgery Workshop, Lanai, Hawaii, March 1995.
187. Elrifai AM, Bailes JE, Taylor MJ, Simon D, Shih SR, Govindan S, Diamond D: Transcranial carotid doppler evaluation of cerebral blood flow velocity in a hemorrhagic shock model of profound hypothermia and complete blood substitution. Ann Meet Am Soc Neuroimaging, San Juan, Puerto Rico, March 1995.
188. Bailes JE: Surgical treatment of stroke. Sharon Regional Health System, Sharon, PA, March 1995.
189. Elrifai AM, Taylor MJ, Bailes JE, Shih SR, Wilberger JE, Maroon JC: A new hypothermic preservation solution for neural tissue preservation. Am Soc Neural Transpl, Tampa, FL, April 1995.
190. Bailes JE: Neurological injuries and the athlete. Amer Assoc Neurol Surgeons. Orlando, FL, April 1995.
191. Bailes JE: Traumatic head and neck injuries. Sports Medicine Update, Dept of Orthopedics, Allegheny General Hospital, Pittsburgh, PA, May 1995.
192. Simon D, Taylor MJ, Elrifai AM, Shih SR, Bailes JE, Davis D, Kluger Y, Diamond D: Profound hypothermia and blood substitution enables resuscitation after hemorrhagic shock. American Society for Artificial Internal Organ Ann Meet, Chicago, IL, May 1995.
193. Bailes JE: Ultraprofound hypothermia. Joint International Congress on Minimally Invasive Techniques in Neurosurgery and Otolaryngology, Pittsburgh, PA, June 1995.
194. Bailes JE: Implementation of telemedicine networks for neurosurgical care. Neurosurgical Society of America, Sea Island, GA, June 1995.
195. Bailes JE: Surgical strategies for giant paraclinoid aneurysms. 4th International Workshop on Cerebrovascular Surgery, Chicago, IL, June 1995.
196. Bailes JE: Future advances in application of blood substitution and ultraprofound hypothermia for cerebrovascular surgery. 4th International Workshop on Cerebrovascular Surgery, Chicago, IL, June 1995.
197. Bailes JE: Stroke prevention and treatment. Armstrong County Memorial Hospital, Kittanning, PA, June 1995.
198. Bailes JE: Current and future prospects of telemedicine systems in neurological surgery. Aequanimitas Annual Meeting, Jackson Hole, WY, July 1995.
199. Bailes JE, Elrifai AM, Taylor MJ, Shih SR, Simon D, Diamond D: Combining ultra-profound hypothermia with blood substitution facilitate resuscitation from hemorrhagic shock. Am Coll Surgeon, Ann Meet, New Orleans, LA, October 1995.
200. Bailes JE: Principles and procedures in neuromonitoring for intracerebral and cerebrovascular surgery. Principles and Applications of Intraoperative Neurophysiologic Monitoring. Pittsburgh, PA November 1995.
201. Bailes JE: Skullbase approaches to intracranial aneurysms. ANI Second Annual

Cerebrovascular Symposium. Pittsburgh, PA December 1995.

202. Dianzumba SB, Bailes JE, Elrifai AM, Shih SR, Emory R, Maroon JC and Reichert N. Intracranial hemorrhage induces cardiac damage. 68th Ann meeting, Am Heart Association, Anaheim, CA, November 1995.
203. Taylor MJ, Bailes JE, Elrifai AM, Shih SR, Simon D, Diamond DL, Maroon JC: Design and evaluation of hypothermic blood substitutes to facilitate resuscitation after hemorrhagic shock and 2 hr of cardiac arrest in canines. NAEMSP, Naples, FL January 1996.
204. Bailes JE: Update on the management of carotid artery disease. AGH Medical Grand Rounds, Pittsburgh, PA, January 1996.
205. Bailes, JE: Design and Evaluation of Hypothermic Blood Substitutes to Facilitate Resuscitation after Neurologic Shock and 2 hr. of Cardiac Arrest in Canines. National Assoc. EMS Physicians, Naples, FL, January 1996 (Finalist-Cerebral Resuscitation Papers)
206. Bailes, JE: Techniques of Carotid Endarterectomy. St. Louis University, Practical Anatomy Workshops, St. Louis, MO, January 1996
207. Bailes, JE: Thrombotic and Embolic Complications of Carotid Endarterectomy. AHA Stroke Meeting, Cerebrovascular Section, San Antonio, TX, January 1996
208. Bailes, JE: Surgical Management of Complex Cerebral Aneurysms. Update on Cerebral Aneurysm Conference. BroMenn Medical Center, Bloomington, Ill., February 1996 (Invited Guest)
209. Bailes, JE: Telemedicine Program in AHERF. Neuroscience Grand Rounds, AGH, Pittsburgh, PA February 1996
210. Bailes, JE: Neurosurgical Management of Head Injuries in Athletes. Conference on Sports Related Concussion, Pittsburgh, March 1996
211. Bailes, JE: Hypothermia Applications in Neurosurgery. International Winter Congress on Minimally Invasive Techniques in Neurosurgery and Otolaryngology, Aspen, CO March 1996.
212. Bailes, JE: Minimally Invasive Techniques Applied to Carotid Artery Surgery. International Winter Congress on Minimally Invasive Techniques in Neurosurgery and Otolaryngology, Aspen, CO, March 1996.
213. Bailes, JE: Assessment and Management of patients with symptomatic and asymptomatic carotid stenosis. New Dimensions in Cardiology for the Primary Care Physician. Pittsburgh, PA March 1996.
214. Bailes, JE: Experimental Hypothermia. 16th Annual Meeting of Japanese Society of Neurological Surgeons. Matsue, Japan, April 1996 (Invited Guest)
215. Bailes, JE: Carotid Endarterectomy. 16th Annual Meeting of Japanese Society of Neurological Surgeons. Matsue, Japan, April 1996 (Invited Guest)
216. Bailes, JE: Thoracolumbar Injuries in Athletes. AANS Annual Meeting. Minneapolis, MN, April 1996.

- 217. Bailes, JE: Brachial Plexus Injuries in Athletes. AGH Sports Medicine Symposium. Pittsburgh, PA April 1996
- 218. Bailes, JE: Overview of Skull Base Procedures in Cerebral Aneurysm Surgery and Moderator, Joint Section of Cerebrovascular Surgery, AANS Annual Meeting, Minneapolis, MN April 1996
- 219. Bailes, JE: Brain attack protocol at AGH. Sharon Regional Health Center, Sharon, PA May 1996.
- 220. Bailes, JE: Design and implementation of a neurosurgical wide area network. Telemedicine 2000, Chicago, IL May 1996.
- 221. Bailes, JE: Assessment of cumulative head injury in professional football players. NFL Players Assoc. Annual Meeting, Albuquerque, NM, April 1996.
- 222. Bailes, JE: Controversial Problems in Athletes with Spinal Abnormalities. Congress of Neurological Surgeons, 46th Annual Meeting, Montreal, Canada, Sept. 1996.
- 223. Bailes, JE: New Therapeutic Strategies for the Management of Head Injuries. Congress of Neurological Surgeons, 46th Annual Meeting, Montreal, Canada, Sept. 1996.
- 224. Dureza C, Bailes JE, Maroon JC: A Preliminary Report on the Use of the Procap in Full Contact Football. Congress of Neurological Surgeons, 46th Annual Meeting, Montreal, Canada, Sept. 1996.
- 225. Dureza C, Fukushima T, Bailes JE, Kiya N, Maroon JC: Minimally Invasive Endoscopic Carotid Endarterectomy. Congress of Neurological Surgeons, 46th Annual Meeting, Montreal, Canada, Sept. 1996.
- 226. Dureza C, Fukushima T, Bailes JE, Levy DI, Maroon JC: The Use of Titanium VCS Autosuture in Cerebrovascular Surgery. Congress of Neurological Surgeons, 46th Annual Meeting, Montreal, Canada, Sept. 1996.
- 227. Bailes JE, Tantuwaya L, Fukushima T: Intraoperative Microvascular Doppler Sonography in Aneurysm Surgery. Congress of Neurological Surgeons, 46th Annual Meeting, Montreal, Canada, Sept. 1996.
- 228. Bailes JE: Construct and deployment of wide area neurosurgical network. National Medical Practice Knowledge Bank Symposium, Pittsburgh, PA Dec. 1996.
- 229. Bailes JE: Complications of carotid artery surgery. Joint Section on Cerebrovascular Surgery Annual Meeting, Anaheim, CA, February 1997.
- 230. Bailes JE: Selected cases of carotid artery origin cerebral ischemia management options. Joint Section on Cerebrovascular Surgery Annual Meeting, Anaheim, CA, February 1997.
- 231. Medary M, Bailes JE: EEG is the most reliable indicator for intraluminal shunting during carotid endarterectomy. Joint Section on Cerebrovascular Surgery Annual Meeting, Anaheim, CA, February 1997.
- 232. Bailes JE: Neurosurgical management of athletic head injuries. Sports related concussion and nervous system injuries. Orlando, FL, February 1997.

- 233. Bailes JE: Overview of athletic spine and spinal cord injuries. Sports related concussion and nervous system injuries. Orlando, FL February 1997.
- 234. Bailes JE: Evolution of telemedicine and application to a national medical knowledge bank. Neuroscience Conference, Allegheny General Hospital, Pittsburgh, PA February 1997.
- 235. Bailes JE: Development and implementation of a neurosurgical wide area network. Implications of an internet based system. Neurosurgical Society of America, Annual Meeting. London, March 1997.
- 236. Bailes JE: Injuries of the cervical spine, spinal cord, and peripheral nerves in athletes, Amer. Assoc. Neurol Surg., Annual Meeting, Denver, CO, April 1997.
- 237. Bailes JE: Head injuries in athletes. American Medical Society for Sports Medicine, Annual Meeting, Colorado Springs, CO, April 1997.
- 238. Bailes JE: Future telemedicine systems in neurosurgery. Southern Neurosurgical Society Ann. Meeting. Pinehurst, NC., May 1997
- 239. Bailes JE: Critical care aspects of athletic nervous system injuries. Congress of Neurological Surgeons Ann. Meeting, New Orleans, LA., Sept. 1997.
- 240. Bailes JE: Anatomical and operative aspects of carotid endarterectomy. Congress of Neurological Surgeons Ann. Meeting, New Orleans, LA., Sept. 1997.
- 241. Bailes JE: Overview and classification of sports medicine for the neurosurgeon. Congress of Neurological Surgeons Ann. Meeting, New Orleans, LA., Sept. 1997.
- 242. Bailes JE: The National Medical Practice Knowledge Bank Project. Congress of Neurological Surgeons Ann. Meeting, New Orleans, LA., Sept. 1997.
- 243. Bailes JE: Computerized systems and Telecommunications: Present and Future Technology. Congress of Neurological Surgeons Ann. Meeting, New Orleans, LA., Sept. 1997.
- 244. Bailes JE: Webmedicine, The Internet and message management for future medicine. Telehealth and the Business of Telemedicine Meeting, Denver, CO., October 1997.
- 245. Bailes JE: Neurological telemedicine, Telemedicine 2000, Orlando, FL., Nov. 1997.
- 246. Bailes JE: Telemedicine systems: applications in trauma. Trauma Tactics Conference, Orlando, FL., Feb. 1998.
- 247. Bailes JE: Organization and function of a county-wide emergency medical services system. Orlando, FL, Jan. 1999
- 248. Bailes JE: Technique and indications for carotid endarterectomy (Session Moderator) Joint Section AANS/CNS and ASITN Stroke Meeting, Orlando, FL., Feb. 1998.
- 249. Bailes JE: Endovascular and surgical therapy for vascular malformations and stroke (Panelist). Joint Section AANS/CNS and ASITN Stroke Meeting, Orlando, FL., Feb. 1998.
- 250. Bailes JE: Chimame "blister" aneurysms. Barrow Neurological Institute Annual Conference,

Phoenix, AZ, March 1998.

251. Bailes JE: Applications of telemedicine systems in neurosurgery. Barrow Neurological Institute Annual Conference, Phoenix, AZ, March 1998.
252. Bailes JE: Strategies for developing a neurosurgical practice partnering with managed care. American Association Neurological Surgery Annual Meeting, Philadelphia, PA, April 1998
253. Bailes JE: Monitoring techniques in carotid endarterectomy. American Association Neurological Surgery Annual Meeting, Philadelphia, PA, April 1998.
254. Bailes JE: Sports Injuries: Special considerations for the head and spine. Indiana Spine Trauma Symposium, Indianapolis, IN, May 1998.
255. Bailes JE: Athletes: Factors in the decision to allow returns to play. . Indiana Spine Trauma Symposium, Indianapolis, IN, May 1998
256. Bailes JE: Applications of telemedicine and the Internet in neurological surgery. American Association Neurological Surgery Annual Meeting, Philadelphia, PA, April 1998.
257. Bailes JE: Developing stroke centers – “Brain Attack” in the community setting. American Association Neurological Surgery Annual Meeting, Philadelphia, PA, April 1998.
258. Bailes JE: Helmet and equipment design for contact sports. American College Sports Medicine. Orlando, FL., June 1998.
259. Elorifai AM, Taylor MJ, Bailes JE, Shih SR et al.: Further development of ultraprofound hypothermia and blood substitution in a canine model with a view to clinical trials. Society of Cryobiology Annual Meeting, Pittsburgh, PA, July 1998.
260. Bailes JE: Use of hypothermia and blood substitution for trauma resuscitation. American College of Surgeons, Annual Meeting, Orlando, FL, October 1998.
261. Bailes JE: Operative treatment of “giant” intracranial aneurysms. American College of Surgeons, Annual Meeting, Orlando, FL., October 1998.
262. Bailes JE: Minor head injuries: incidence, pathophysiology, and treatment. American College of Surgeons, Annual Meeting, Orlando, FL, October 1998.
263. Bailes JE: Management of athletic head and spinal injuries. American Association of Neurological Surgeons, Annual Meeting, Seattle, WA, October 1998.
264. Bailes JE: Current technologies for surgical practices. American Association of Neurological Surgeons Annual Meeting, Seattle, WA, October 1998.
265. Bailes JE: Internet and its applications in medicine and neurosurgery. American Association of Neurological Surgeons Annual Meeting, Seattle, WA, October 1998.
266. Bailes JE: Neurosurgical evaluation and treatment of brain and spinal injuries. Medical Management Group of Orlando. Orlando, FL, Jan. 1999.
267. Bailes JE: Carotid endarterectomy and stenting. Joint meeting of Section of Cerebrovascular

Surgery and ASITN. Nashville, TN Feb. 1999.

- 268. Bailes JE: When to play following athletic central nervous system injury. 23rd Annual Internal Medicine Conference, Orlando, FL, Mar. 1999
- 269. Bailes JE: Issues in management of injured athletes. American Association of Neurological Surgeons Annual Meeting, New Orleans, LA, April 1999.
- 270. Bailes JE: Issues in management of injured athletes. American Association of Neurological Surgeons Annual Meeting, New Orleans, LA, April 1999.
- 271. Bailes JE: Establishing a modern medical practice utilizing networking and high technology. American Association of Neurological Surgeons Annual Meeting, New Orleans, LA, April 1999
- 272. Bailes JE: Head injuries in athletes. Spinal Symposium on Athletic Injuries. American Association of Neurological Surgeons Annual Meeting, New Orleans, LA, April 1999
- 273. Bailes JE: Management of athletic head injuries. Sports related nervous system injuries annual meeting, Orlando FL May 1999
- 274. Bailes JE: Management of spinal injuries in athletes, Sports related nervous system injuries annual meeting, Orlando FL May 1999
- 275. Bailes JE: Head and spinal injuries in sports. Florida Athletic Trainers Association Annual Meeting. Orlando, FL. June 1999
- 276. Bailes JE: Surgical treatment of stroke. St. Luke's Hospital Annual Stroke Meeting. Lake Geneva, WI Sept. 1999
- 277. Bailes JE: Surgical treatment for cerebral aneurysms and AVM's. St. Luke's Hospital Annual Stroke Meeting. Lake Geneva, WI Sept. 1999.
- 278. Bailes JE: Teleradiology and EMS in future treatment of the stroke patient. St. Luke's Hospital Annual Stroke Meeting. Lake Geneva, WI Sept. 1999.
- 279. Bailes JE: Concussion in sports. Congress Neurological Surgeons Annual Meeting. Boston, MA Nov. 1999.
- 280. Bailes JE: The neurosurgeon's role in future EMS systems. Congress of Neurological Surgeons Annual Meeting, Boston, MA, November 1999
- 281. Bailes JE: Head injuries in sports, Congress of Neurological Surgeons Annual Meeting. Boston, MA, November 1999.
- 282. Bailes, JE: Development and structure of EMS systems for stroke and neurotrauma. Gateway to the Brain Conference. Orlando Science Center, Orlando, FL, November 1999.
- 283. Bailes JE: Carotid endarterectomy. Indications and results. Joint Meeting ASITN and JSCVS. New Orleans, LA. February 2000.
- 284. Bailes JE: Symposium moderator. Skull base meningiomas. North American Skull Base Society. Phoenix, AZ. February 2000.

- 285. Bailes JE: Concussion management in athletes. American Association Neurological Surgeons Annual Meeting. San Francisco, CA. April 2000.
- 286. Bailes JE: Concussion in athletes-classification systems. American Assoc Neurological Surgeons Annual Meeting. San Francisco, CA. April 2000.
- 287. Bailes JE, Jordan BD: Concussion history and current neurological symptoms among retired professional football players. American Academy of Neurology Annual Meeting. San Diego, CA. May 2000
- 288. Bailes JE: Management of concussion in athletes. Neurosciences Teaching Weekend. Morgantown, West Virginia. September 2000
- 289. Bailes JE: Surgical treatment of stroke. Neurosciences Teaching Weekend. Morgantown, West Virginia. September 2000
- 290. Bailes JE: Surgical treatment of stroke. Hal Wanger Family Medicine Conference. Morgantown, West Virginia. October 2000
- 291. Sadrolhefazi A, Miele V, Carr A, Bailes JE: A Retrospective Review of Neurological Injuries Related to Sports and Recreational Activities. American Association Neurological Surgeons Annual Meeting Toronto 2001
- 292. Sadrolhefazi A, Miele V, Carr A, Bailes JE: A Retrospective Review of Neurological Injuries Related to Sports and Recreational Activities. The Neurological Society of the Virginias Annual Meeting, Homestead, VA, January 2001.
- 293. Bailes JE: Intrathecal thrombolysis for aneurismal intraventricular hemorrhage. The Neurological Society of the Virginias Annual Meeting, Homestead, VA, January 2001.
- 294. Carr A, Miele V, Bailes JE: Neurologic Hunting Injuries. Neurotrauma and Sports Medicine for the New Millennium Conference, Park City, UT. March 2001.
- 295. Bailes JE: Spinal stenosis in the athlete. American Association of Neurological Surgeons Annual Meeting. Toronto, April 2001.
- 296. Bailes JE: Skullbase approaches for aneurysms of the vertebrobasilar circulation. Duke University, International Neurosciences Symposium, Raleigh, NC. April 2001.
- 297. Miele V, Sadrolhefazi A, Carr A, Bailes JE: A Retrospective Review of Neurological Injuries Related to Sports and Recreational Activities. Edgar F. Heiskell Memorial Trauma Conference, Morgantown WV. September 2001.
- 298. Carr A, Miele V, Bailes JE, et al: Factors that Influence Neurologic Injuries and Death in ATV Accidents: a Ten Year Retrospective Review at the Jon Michael Moore Trauma Center. Edgar F. Heiskell Memorial Trauma Conference, Morgantown WV. September 2001.
- 299. Bailes JE, Miele V, Voelker J: Boxing and the neurosurgeon. Congress of Neurological Surgeons, San Diego, CA. September 2001.
- 300. Sadrolhefazi A, Miele V, Bailes JE: Influence of Head Position on the Effectiveness of Twist Drill Craniostomy for Chronic Subdural Hematoma. Congress of Neurological Surgeons, San Diego, CA. September 2001.

301. Bailes J, Day A, Miele V: Normal Perfusion Pressure Breakthrough in Small Arteriovenous Malformations. Congress of Neurological Surgeons, San Diego, CA. September 2001.
302. Bailes JE: Emerging surgical indications for stroke. Neurosciences Teaching Weekend, Morgantown, WV. September 2001
303. Bailes JE: Prehospital care and secondary injury. Presentation for head trauma. Ohio Valley Trauma Update, Wheeling, WV. October 2001.
304. Bailes JE: Intrathecal Thrombolysis for Aneurysmal Intraventricular Hemorrhage. American Academy of Neurological Surgery Annual Meeting. Palm Beach, FL. November 2001.
305. Bailes JE: The medical database for effects of chronic head injury in NFL athletes. Center for the Study of Retired Athletes. Chapel Hill, NC. November 2001.
306. Bailes JE: Management of difficult cerebral aneurysms. Allegheny General Hospital, Pittsburgh, PA. December 2001.
307. Bailes JE: Neurosurgery. Preston Memorial Hospital, Kingwood, WV. December 2001.
308. Bailes JE, Carson LV, Rosen CL: Neurosurgery, Doctors On Call program, broadcast by WVPTV. December 2001.
309. Bailes JE, Miele VJ, Unkelbach MH, Medary M, Voelker JL: Improved outcomes in poor-grade aneurysm patients. Neurological Society of the Virginias meeting, The Homestead, VA, January 2002.
310. Sadrolhefazi A, Miele V, Bailes JE: Influence of Head Position on the Effectiveness of Twist Drill Craniostomy for Chronic Subdural Hematoma. Neurological Society of the Virginias meeting, The Homestead, VA, January 2002.
311. Wilkinson C, Miele V, Nestor S, Rosen C, Bailes JE: Phenytoin and Magnesium Sulfate as Neuroprotective Agents in Acute Spinal Cord Injury. Neurological Society of the Virginias meeting, The Homestead, VA, January 2002.
312. Carr A, Miele V, Bailes JE, Mucha P, Helmkamp J: Factors that Influence Neurologic Injuries and Death in ATV Accidents. Neurological Society of the Virginias meeting, The Homestead, VA, January 2002.
313. Miele V, Hall L, Unkelbach M, Sadrolhefazi A, Carr A, Bailes JE: Soccer Related Neurological Injuries. Neurological Society of the Virginias meeting, The Homestead, VA, January 2002.
314. Bailes JE: Surgical Treatment of Strokes. Fairmont General Hospital, Fairmont, WV. February 2002.
315. Bailes JE: Boxing and the neurosurgeon. Neurotrauma and Sports Medicine Review, Orlando, FL. February 2002
316. Bailes JE: Adult spine and spinal cord injuries: return to play issues. Neurotrauma and Sports Medicine Review, Orlando, FL. February 2002

- 317. Bailes JE: Overview of Neurosurgery. 1st & 2nd year Medical Students February 2002.
- 318. Bailes JE: Head Injuries and Return to Play Issues. Graduate level Athletic Trainers class, West Virginia University, Morgantown, WV. March 2002.
- 319. Bailes JE, Miele VJ, Unkelbach MH, Medary M, Voelker JL: Improved outcomes in poor-grade aneurysm patients. Poster presentation, AANS Annual Meeting, Chicago, IL, April 2002.
- 320. Miele VJ, Price K, Pryputniewicz D, Becker D, Bailes JE: Analysis of Knockouts and Fatalities in the Sport of Professional Boxing. Poster presentation, AANS Annual Meeting, Chicago, IL, April 2002.
- 321. Miele V, Hall L, Unkelbach M, Sadrolhefazi A, Carr A, Bailes JE: Soccer Related Neurological Injuries. AANS Annual Meeting, Chicago, IL, April 2002.
- 322. Carr A, Miele V, Bailes JE, Mucha P, Helmkamp J: Factors that Influence Neurologic Injuries and Death in ATV Accidents. AANS Annual Meeting, Chicago, IL, April 2002.
- 323. Bailes JE, Guskiewicz K, Marshall S: Recurrent Sports-Related Concussion Linked to Clinical Depression. AANS Annual Meeting, Chicago, IL, April 2002.
- 324. Wilkinson C, Miele V, Nestor S, Rosen C, Bailes JE: Phenytoin and Magnesium Sulfate as Neuroprotective Agents in Acute Spinal Cord Injury. AANS Annual Meeting, Chicago, IL, April 2002.
- 325. Bailes JE: What is a Gamma Knife? WVUH Leadership Forum (Managers & Supervisors) Morgantown, WV. April 2002.
- 326. Bailes JE: Vasospasm. ICU In-service, Morgantown, WV. April 2002.
- 327. Bailes JE: Skull Base Approaches to Basilar Aneurysms. International Skull Base Symposium, West Palm Beach, FL. May 2002.
- 328. Bailes JE: Microvascular doppler monitoring during cerebral aneurysms. American Society of Neurophysiological Monitoring Annual Conference, Orlando, FL. May 2002.
- 329. Bailes JE: Stroke. MS 1 class, WVU School of Medicine, Morgantown, WV. May 2002.
- 330. Bailes JE: Neurological consequences of a professional football career. NFL Retired Players' Convention, Phoenix, AZ. May 2002.
- 331. Bailes JE: Vascular concerns. Surgery Clerkship lecture, WVU School of Medicine, Morgantown, WV. June 2002.
- 332. Bailes JE: Neurosurgical Management of the Athlete: the Spectrum of Traumatic Brain Injury in Athletes. New Developments in Sports-Related Concussion, UPMC Conference, Pittsburgh, PA. July 2002.
- 333. Bailes JE: Tactical Medicine. WV State Police, Charleston, WV. July 2002.
- 334. Bailes JE: Heat Stroke & Dietary Supplements. Aequanimitas Society meeting, Cleveland, OH. August 2002.

- 335. Bailes JE: Tactical Medicine. WV National Guard, Kingwood, WV. August 2002.
- 336. Bailes JE: Management of Head & Neck Injuries in Athletes. ACOS Meeting, Orlando, FL. Sept. 2002
- 337. Bailes JE: Management of Difficult Cerebral Aneurysms. ACOS Meeting, Orlando, FL. Sept. 2002
- 338. Bailes JE: CNS Meeting, Philadelphia, PA Sept. 2002
- 339. Bailes JE: The Role of Gamma Knife in the Neurosurgeon's Armamentarium. Gamma Knife Open House, Morgantown, WV. November 2002.
- 340. Bailes JE: Medical Perspectives on Bioterrorism. Dept. of Justice. Alexandria, VA. December 2002.
- 341. Bailes JE: Concussion. Neurotrauma and Sports Medicine Conference. Orlando, FL. February 2003.
- 342. Bailes JE, Miele V: Fatalities in Organized Sports: What Have We Learned? AANS Meeting, San Diego, CA. April/May 2003.
- 343. Miele V, Price K, Pryputniewicz D, Becker D, Bailes JE: Analysis of Knockouts and Fatalities in the Sport of Professional Boxing. AANS Meeting, San Diego, CA. April/May 2003.
- 344. Bailes JE: Recurrent Sport-Related Concussion Linked to Clinical Depression. AANS Meeting, San Diego, CA. May 2003.
- 345. Bailes JE: Challenges in Identifying & Treating Sports Injuries. AANS Meeting, San Diego, CA. May 2003.
- 346. Bailes JE: Concussions in the NFL. K. Douglas Bowers Orthopedic Lectureship, Morgantown, WV. September 2003.
- 347. Bailes JE: Gamma Knife: Radiosurgery in the Treatment of Brain Tumors. Fall Cancer Conference, Morgantown, WV. October 2003.
- 348. Bailes JE: Telemedicine. International and Virtual Neurological Surgery Colloquium, CNS Meeting, Denver, CO. October 2003.
- 349. Bailes JE: The Science of Sports Medicine. CNS Meeting, Denver, CO. October 2003.
- 350. Miele V, Bailes JE: Neurological Injuries to Occupants riding in the Cargo Area of Pickup Trucks in West Virginia. CNS Meeting, Denver, CO. October 2003.
- 351. Miele V, Becker D, Bailes JE: Death in the Ring – Analysis of 10 Boxing Fatalities. CNS Meeting, Denver, CO. October 2003.
- 352. Miele V, Carson L, Bailes JE: Acute Subdural Hematoma in a Female Boxer: Case report and a Discussion of Unique Risks to the Female Participant. CNS Meeting, Denver, CO. October 2003.
- 353. Bailes JE: Surgical Treatment of Difficult and Complex Aneurysms. 3rd Annual Temporal Bone

- Dissection Course and International Symposium on Clinical Neurosciences and Cerebrovascular Skull Base Surgery. Morgantown, WV. October 2003.
354. Bailes JE: Sideline evaluation and treatment of closed head injury. Family Practice and Sports Medicine Conference. Huntington, WV. November 2003.
355. Bailes JE: Neurosurgical perspective on acute ischemic stroke. AANS Cerebrovascular Section Meeting. San Diego, CA. February 2004.
356. Bailes JE: Periprocedural neuroprotection. AANS Cerebrovascular Section Meeting. San Diego, CA. February 2004.
357. Bailes JE: Current tenets in management in athletic head injuries. WV Trauma Symposium. Canaan Valley, WV. February 2004.
358. Bailes JE: Neurosurgical sports injuries. Synthes Maxillofacial Annual Meeting. Snowbird, UT. March 2004.
359. Bailes JE: Contemporary management in neurological sports medicine. J Jay Keegan Memorial Lectureship, University of Nebraska. Omaha, NE. April 2004.
360. Bailes JE: Neurosurgical athletic brain injuries. 2004 Sports Concussion and Spine Injury Conference. Boston, MA. May 2004.
361. Bailes JE: Temporary paralysis in athletes. Neurosurgical Society of America, Annual Meeting, Santa Fe, NM. June 2004.
362. Bailes JE: Boxing/Martial Arts and Concussion. New Developments in Sports-Related Concussion Conference, UPMC, Pittsburgh, PA. July 2004.
363. Bailes JE: Head Injury. Emergency/Trauma Symposium 2004, Wheeling, WV. October 2004.
364. Bailes JE: CT perfusion studies to define time window for stroke interventions. Neurosurgical Society of the Virginias annual meeting. The Greenbrier, WV. January 2005.
365. Bailes JE: Management of severe head injury and intracranial pressure. WV Trauma Symposium, Canaan Valley, WV. February 2005.
366. Bailes JE: Management of difficult cervical spine injuries in athletes. Neurotrauma Symposium, Orlando, FL. March 2005.
367. Bailes JE: On-field management of the injured athlete. Neurotrauma Symposium, Orlando, FL. March 2005.
368. Bailes JE: NATA/AFCA Spearing in football task force recommendations. Sports Related Concussion and Spine Injury Conference, Boston. May 2005.
369. Bailes JE: CT perfusion protocol for stroke. Aequanimitas Meeting, Seagrove Beach, FL. June 2005.
370. Bailes JE: West Virginia: Electronic medical records. West Virginia Healthcare Summit 2005. The Greenbrier, Lewisburg, WV. August 2005.

- 371. Bailes JE: Electronic health records initiative in West Virginia. Electronic Health Records Initiative Committee, Stonewall Resort, Roanoke, WV. September 2005.
- 372. Bailes JE: Electronic medical records. West Virginia Health Information Management Association annual meeting. Flatwoods, WV. September 2005.
- 373. Bailes JE: Cervical disease. WVUH Spine Conference. Lakeview Resort, Morgantown, WV. October 2005.
- 374. Bailes JE: Steroids in sports. Vital Signs television show, Charleston, WV. November 2005.
- 375. Bailes JE: Giant aneurysms panel discussion. Extracranial and Intracranial Bypass workshop, Barrow Neurological Institute, Phoenix, AZ. February 2006.
- 376. Bailes JE: Concussion and brain injury: treatment with high dose DHA. Management of the Neurotrauma Patient, Toronto, ON. April 2006.
- 377. Bailes JE: Electronic Medical Records. Ohio Valley Medical Center, Wheeling, WV. May 2006.
- 378. Bailes JE: Stroke. State of the Stroke West Virginia meeting, Charleston, WV. September 2006.
- 379. Bailes JE: Head injuries in the elderly. OVMC Trauma Symposium. Wheeling, WV. October 2006.
- 380. Bailes JE: E-Health and the effect on rural America. WV Rural Health Conference, Stonewall Resort, Roanoke, WV. October 2006.
- 381. Bailes JE: Sports injury update. Southern Neurosurgical Society Annual Meeting, Sea Island, GA. March 2007.
- 382. Bailes JE: Importance of HER. National Technology Transfer Center, Wheeling, WV. March 2007.
- 383. Bailes JE: Argument to continue boxing. AANS, Washington, DC. April 2007.
- 384. Bailes JE: Spectrum of outcome of sports concussion. The National Concussion Summit, Marina Del Rey, CA. April 2007.
- 385. Bailes JE: Concussion in sports: state of the science. Moderator, panel discussion. The National Concussion Summit, Marina Del Rey, CA. April 2007.
- 386. Bailes JE: Long Term Cognitive Impairment in the NFL Player. Sports Related Conference on Concussion & Spine Injury, Boston, MA. April 2007.
- 387. Bailes JE, Lovell M: Other studies of retired players. NFL Concussion Summit, Chicago, IL. June 2007.
- 388. Bailes JE, Maroon JC, Casson I: Does concussion lead to pugilistic dementia and alzheimers? NFL Concussion Summit, Chicago, IL. June 2007.
- 389. Bailes JE: Drug testing, panel discussion. WVU Sports Law Symposium. Morgantown, WV. October 2007.
- 390. Bailes JE: The neurosurgeon's livelihood in sports medicine. 2007 Annual Clinical Assembly of

Osteopathic Specialists. Las Vegas, NV. October 2007.

- 391. Bailes JE: Is the hassle of setting up and using telemedicine worth the return? 2007 Annual Clinical Assembly of Osteopathic Specialists. Las Vegas, NV. October 2007.
- 392. Bailes JE: Having long-term results with complex aneurysms: what do I do now? 2007 Annual Clinical Assembly of Osteopathic Specialists. Las Vegas, NV. October 2007.
- 393. Bailes JE: Tauopathy dementia in the spectrum of athletic brain injury. Neurosurgical Society of the Virginias annual meeting. The Greenbrier, WV. January 2008.
- 394. Bailes JE: Neurosurgical trauma. NYUSOM Neurosurgery Grand Rounds. New York, NY. March 2008.
- 395. Bailes JE: NASA, Houston, TX. March 2008.
- 396. Bailes JE, Roberts L: Doctoring through a media frenzy. AMA Medical Communications Conference, San Diego, CA. April 2008.
- 397. Bailes JE: Life after sports: the challenges of informed decision making. Sports Concussion Seminar, Marina Del Rey, CA. April 2008.
- 398. Handley K, Bailes JE: DHA – the important omega 3 fatty acid in brain structure: its relevance in athletic brain injury. Sports Concussion Seminar, Marina Del Rey, CA. April 2008.
- 399. Bailes JE: Chronic traumatic encephalopathy in the professional athlete. Sports Medicine Conference, Boston, MA. May 2008.
- 400. Bailes JE, Guskiewicz K, Cantu R, et al: Brain injury panel discussion. Sports Medicine Conference, Boston, MA. May 2008.
- 401. Bailes JE: Concussion. Athletic Trainers Conference. UGa, Athens, GA. May 2008.
- 402. Sears B, Bailes JE: Omega 3 fatty acid supplementation reduces the extent of axon damage after brain trauma. ISSAFL 2008, Kansas City, MO. May 2008.
- 403. Bailes JE: Improving communication during a public health emergency. HLS Conference, Morgantown, WV. June 2008.
- 404. Bailes JE: Omega 3 EFA and head injury. Zone Labs, Bermuda. July 2008.
- 405. Bailes JE: Are there long-term neuropathological changes in athletes. New Development in Sports-Related Concussion Conference. Pittsburgh, PA. July 2008.
- 406. Bailes JE, Omalu B: Human and experimental evidence of neurodegeneration in contact sports. American Academy of Neurological Surgeons annual meeting, Phoenix, AZ. September 2008.
- 407. Bailes JE: Is there potential for long-term effects of athletic concussion? Visiting professor, Wayne State University Dept of Neurosurgery. Detroit, MI. October 2008.
- 408. Bailes JE: Use of Omega 3 fatty acids in neural trauma. 2nd International Zone Conference, Anti-inflammatory Medicine. Cancun, Mexico. November 2008.

- 409. Bailes JE: Concussion. American Football Coaches Association annual meeting. Nashville, TN. January 2009.
- 410. Bailes JE: Long-term consequences of athletic mild traumatic brain injury. Interurban Neurosurgical Society annual meeting. Chicago, IL. March 2009.
- 411. Bailes JE: Permanent brain injury – possibly to be caused by contact sports. Grand rounds, University of Illinois Chicago, IL. June 2009.
- 412. Bailes JE: New understanding of causes and consequences of sport-related concussion. Annual meeting Neurosurgical Society of the Virginias, The Greenbrier, WV. January 2010.
- 413. Bailes JE: Concussion 2010 – brain injury and brain armor. Big Sky Athletic Training and Sports Medicine Conference, Big Sky, MT. February 2010.
- 414. Bailes JE: Latest and greatest in traumatic brain injury. WV Trauma Symposium, Roanoke, WV. February 2010.
- 415. Bailes JE: What is chronic traumatic encephalopathy? Cyril H. Wecht Institute of Forensic Science and Law, Concussion Debate, Duquesne University, Pittsburgh, PA. March 2010.
- 416. Bailes JE: Controversies in the management of TBI in professional athletes. AANS, Philadelphia, PA. May 2010.
- 417. Bailes JE: Traumatic brain injury and concussion. Neurological and Neurosurgical Therapeutics 2010: Contemporary Diagnosis and Management. University of South Florida, Tampa, FL. May 2010.
- 418. Bailes JE: Spinal injuries in athletes. WVU Spine Conference, Morgantown, WV. September 2010.
- 419. Bailes JE: Head traumas and concussions. Dieter-Porter Medical Lecture, Washington & Jefferson College, Washington, PA. October 2010.
- 420. Bailes JE: Mining disasters and safety: the Sago Mine experience. Grand Rounds, Allegheny General Hospital, Pittsburgh, PA. December 2010
- 421. Bailes JE: Reduction of Concussion and Subconcussive Traumatic Brain Injury Through SLOSH Mitigation. Annual meeting Neurosurgical Society of the Virginias, The Greenbrier, WV. January 2011.
- 422. Bailes JE, Maroon JC, Raksin PB: Football injuries and concussion: assessment, return to play, long-term sequelae and the neurosurgeon's role. CNS University of Neurosurgery Trauma Webinar. January 2011.
- 423. Bailes JE: The evolution in our knowledge of concussions: state of the art in 2011. WVATA Annual Sports Medicine Conference, Morgantown, WV. February 2011.
- 424. Bailes JE: Mild traumatic brain injuries in athletes: are they really mild? Dept. of Neurosurgery, Stanford University, Stanford, CA. March 2011.

- 425. Bailes JE: Mild traumatic brain injuries in athletes: are they really mild? Sally Herrington Goldwater Visiting Professor, Barrow Neurological Institute, Phoenix, AZ. March 2011.
- 426. Bailes JE: Sports-related traumatic brain injury. AANS, Denver, CO. April 2011.
- 427. Bailes JE: Subconcussion impacts: their role in producing long term brain damage. AANS, Denver, CO. April 2011.
- 428. Bailes JE: DHA and the research showing its benefits for the treatment and prevention of head concussions. Collegiate Strength and Conditioning Coaches Association National Conference, Kansas City, MO. May 2011.
- 429. Bailes JE: Subconcussive blows: what are their consequences in sport? Annual National Summit on Sports Concussion, Los Angeles, CA. May 2011.
- 430. Bailes JE: Repeated sub-concussive blows: do they occur and what do we know? National Athletic Trainers Association annual meeting, New Orleans, LA. June 2011.
- 431. Bailes JE: Cumulative brain damage after sports concussion. National Neurotrauma Symposium, Fort Lauderdale, FL. July 2011.
- 432. Bailes JE: Perioperative complications following SAH. University of Miami Aneurysm Workshop. Miami, FL Nov 2011
- 433. Bailes JE: Mild traumatic brain injuries in athletes: are they really mild? Georgia Neurosurgical Society annual meeting. Atlanta, GA. Dec 2011
- 434. Bailes JE: Brain protection internally by slosh mitigation. Georgia Neurosurgical Society annual meeting. Atlanta, GA Dec 2011.
- 435. Bailes JE: Mild traumatic brain injuries in athletes: are they really mild? NorthShore University HealthSystem, Medical Grand Rounds. February 6, 2012
- 436. Bailes JE: Cervical Spine Injuries in Athletes. Dept. of Orthopedics, NorthShore University HealthSystem, Evanston, IL. April 6, 2012
- 437. Bailes JE: Risks for CTE. Dept. of Neurosurgery, Medical College of Wisconsin. May 18, 2012
- 438. Bailes JE: Mild traumatic brain injuries in athletes: are they really mild? 2012 Annual Clinical Assembly of Osteopathic Surgeons. Chicago, IL October 1, 2012
- 439. Bailes JE: Return to Play Issues in Sports-Related Neurotrauma. American College of Surgeons Clinical Congress, Chicago, IL. October 2, 2012
- 440. Bailes JE: Mild traumatic brain injuries in athletes: are they really mild? 6th Annual Fall CME Meeting - Association of Neurosurgery Physician Assistants. Chicago, IL October 6, 2012
- 441. Bailes JE: Treating Brain Tumors — A Collaborative Approach to Care. Understanding Cancer Conference, Evanston Hospital, Evanston, IL Dec. 3, 2012
- 442. Bailes JE: Mild traumatic brain injuries in athletes: are they really mild? The Epilepsy Foundation of Greater Chicago, Evanston, IL February 23, 2013

- 443. Bailes JE: Concussion in football: An Update. Andrews Institute "Injuries in Football Conference, Destin, FL. March 2, 2013
- 444. Bailes JE: Concussion management: Best practices for physician diagnosis. NCAA Concussion Task Force, Indianapolis, IN. April 12, 2013
- 445. Bailes JE: Concussion management: Role in concussion management. NCAA Concussion Task Force, Indianapolis, IN. April 12, 2013
- 446. Bailes JE: Chronic traumatic encephalopathy: Concussion cause vs. concussion correlation. NCAA Concussion Task Force, Indianapolis, IN. April 12, 2013
- 447. Bailes JE: Chronic Traumatic encephalopathy: Risks and predispositions. NCAA Concussion Task Force, Indianapolis, IN. April 12, 2013
- 448. Bailes JE: Chronic traumatic encephalopathy: Numerator and denominator-population estimates. NCAA Concussion Task Force, Indianapolis, IN. April 12, 2013
- 449. Bailes JE: Practice guidelines and impact on concussion. NCAA Concussion Task Force, Indianapolis, IN. April 12, 2013
- 450. Bailes JE: Mild traumatic brain injuries in athletes: Are they really mild? Keynote address at the American Medical Society for Sports Medicine, San Diego, CA. April 18, 2013
- 451. Bailes JE: Consequences: Avoidance & managements of concussion & sports Injury. Practical Clinic 037 Concussion & Sports Injury: State of the Art. American Association of Neurological Surgeons Annual Scientific Meeting, Saturday, April 27, 2013, New Orleans, LA
- 452. Bailes JE: Historical contributions of neurosurgeons to neurological sports medicine. American Association of Neurological Surgeons Annual Meeting, Saturday, April 30, 2013, New Orleans, LA
- 453. Bailes JE: An Update on premorbid diagnosis of CTE. It's now a reality, but how will it help? 7th Annual National Summit on Sports Concussion. May 10, 2013, Atlanta, GA.
- 454. Bailes JE: Subconcussive blows and risk of CTE. 7th Annual National Summit on Sports Concussion. May 10, 2013, Atlanta, GA.
- 455. Bailes JE: Mild traumatic brain injuries in athletes: Are they really mild? Vivian L. Smith Department of Neurosurgery Grand Rounds, University of Texas Medical School at Houston. October 3, 2013.
- 456. Bailes JE: Concussion – Much ado about nothing? Congress of Neurological Surgery Annual Meeting. October 19, 2013, San Francisco, CA
- 457. Bailes JE: Frequency, subconcussion: It is real and what is its impact. Congress of Neurological Surgery Annual Meeting. October 21, 2013, San Francisco, CA
- 458. Bailes JE: Frequency, magnitude, and distribution of head impacts in Pop Warner Football. Congress of Neurological Surgery Annual Meeting. October 23 2013, San Francisco, CA
- 459. Bailes JE: Clinical Management of Concussions. New York Neurosurgery at A Cushing

Neuroscience Institute Symposium Focus on Brain Tumor, Cerebrovascular and Head Trauma.
December 14, 2013, New York City, NY

- 460. Bailes JE: Subconcussion and Its Effects. NCAA Safety in College Football Summit. January 22, 2014, Atlanta, GA
- 461. Bailes JE: Effect of rule changes on mTBI in youth football. NCAA Safety in College Football Summit. January 22, 2014, Atlanta, GA
- 462. Bailes JE: Long term consequences of repetitive brain trauma: CTE and Neurodegeneration. United Nations Headquarters. January 29, 2014, New York, NY
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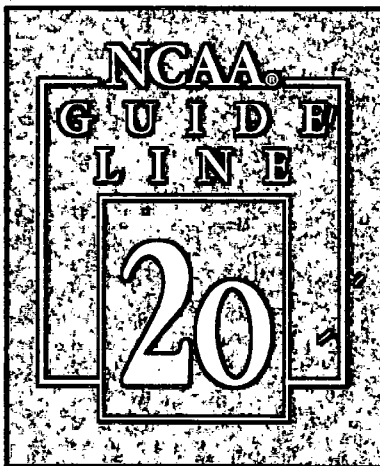
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BAILES EXHIBIT 2

1994-95 NCAA[®] Sports Medicine Handbook





Concussion and Second-Impact Syndrome

June 1994

Concussion and second-impact syndrome are two potentially life-threatening risks to which student-athletes are exposed. It is estimated that concussions are suffered by one in five high-school football players each season, which, if accurate, means that more than 250,000 concussions occur annually at that level alone. One might assume similar risk of injury to college football players. In college football, nine of 10 head injuries are reported to be concussions, with football head injuries occurring twice as frequently as neck injuries.

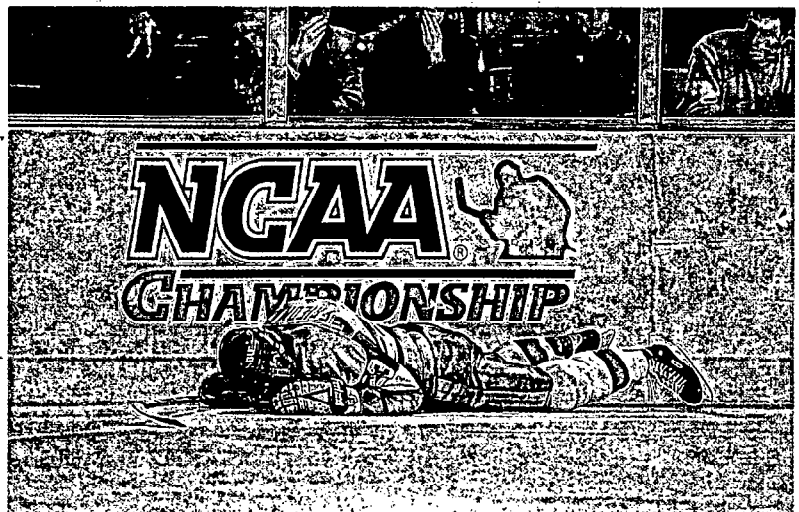
Some of the mild concussions, the so-called "bell rung" or "ding," with no loss of consciousness or

posttraumatic amnesia may go unrecognized by the coaches, athletics trainers, fellow players or team physicians. The cumulative effect of these mild concussions is unknown, so guidelines for sports participation are difficult to establish. Furthermore, categorizations for severity of concussion vary and are not universally accepted, compounding the difficulty of establishing guidelines for participation.

The Colorado Medical Society Sports Medicine Committee has developed basic guidelines for the management of concussion in sports. These guidelines, summarized in the following table, have reasonable application to clear-

ance guidelines in the preparticipation evaluation. Although these guidelines may assist in clinical decision-making, they are not absolute and should not be substituted for the clinical judgment of the examining physician. If there are any questions as to the severity of past head trauma, or if the trauma required intracranial surgery, clearance should be deferred until further records are obtained and/or neurosurgical evaluation is performed. No athlete should be allowed to return to contact sports on the same day that a grade-three concussion was received.

A table of sideline evaluation for possible concussion also is included for reference.





Guideline 2-O Continued

Concussion and Second-Impact Syndrome

Grading Concussions in Sports and Guidelines for Return to Play*

*—These guidelines are not absolute and therefore should not substitute for the clinical judgment of the examining physician.

Grading		Guidelines		
Severity	Signs/symptoms	First concussion	Second concussion	Third concussion
Grade I (mild)	Confusion without amnesia; no loss of consciousness	May return to play if asymptomatic ^{††} at least 20 minutes	Terminate contest/practice; may return to play if asymptomatic ^{††} for at least one week	Terminate season; may return to play in three months if asymptomatic ^{††}
Grade II (moderate)	Confusion with amnesia [†] ; no loss of consciousness [†]	Terminate contest/practice; may return to play if asymptomatic ^{††} for at least one week	Consider terminating season, but may return to play if asymptomatic ^{††} for one month	Terminate season; may return to play next season if asymptomatic ^{††}
Grade III (severe)	Loss of consciousness [†]	Terminate contest/practice and transport to hospital; may return to play one month after two consecutive asymptomatic ^{††} weeks; conditioning allowed after one asymptomatic ^{††} week	Terminate season; may return to play next season if asymptomatic ^{††}	Terminate season; strongly discourage return to contact/collision sports

*—Posttraumatic amnesia (amnesia for events following the impact) or more severe retrograde amnesia (amnesia for events preceding the impact).

†—Some clinicians include “brief” loss of consciousness in Grade II and reserve “prolonged” loss of consciousness for Grade III. However, the definitions of “brief” and “prolonged” are not universally accepted.

††—No headache, confusion, dizziness, impaired orientation, impaired concentration or memory dysfunction during rest or exertion. (Adapted from: Colorado Medical Society. Report of the Sports Medicine Committee: Guidelines for the management of concussion in sports (revised). Denver: Colorado Medical Society, 1991.)

Table courtesy of:

Preparticipation physical evaluation (monograph). Kansas City, Missouri: American Academy of Family Physicians, American Academy of Pediatrics, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, American Osteopathic Academy of Sports Medicine, 1992.



Guideline 2-0 Continued

Concussion and Second-Impact Syndrome

Sideline Evaluation For Concussion
(from Colorado Head Injury Foundation, Inc.)

1. Mental-Status Testing

a. Orientation: Time, place, person and situation (circumstances of injury)

b. Concentration: Digits Backward

3-1-7

4-6-8-2

5-9-3-7-4

Months of year in reverse order

c. Memory: Names of teams in prior contest

President, Governor, Mayor.

Recent newsworthy events,

3 words and 3 objects at 0 and 5 minutes.

Details of contest (plays, moves, strategies, etc.)

2. Exertional Provocative Tests:

40-yard sprint

5 push-ups

5 sit-ups

5 knee bends

(Any appearance of associated symptoms is abnormal, e.g., headache, dizziness, nausea, unsteadiness, photophobia, blurred or double vision, emotional lability or mental-status changes.)

3. Neurological Tests:

Pupils:

Symmetry and reaction

Coordination:

Finger-nose finger and tandem

Sensation:

Finger-nose (eyes closed) and Romberg



Guideline 2-O Continued

Concussion and Second-Impact Syndrome

Second-Impact Syndrome

The medical staff also needs to be aware of the often lethal consequences of the second-impact syndrome that occur when an individual sustains a second, often minor, blow to the head before the initial symptoms of the head injury are over. The resulting loss of autoregulation of the brain's blood supply could result in vascular engorgement and herniation of the lower brain, resulting in death. There is an approximately 50 percent mortality rate associated with second-impact syndrome. Treatment is prompt intubation, hyperventilation and IV osmotic diuretics.

Summary

The attending medical staff should not allow a player to resume participation in sports until the injured student-athlete has fully recovered from his/her postconcussive symptoms. With regard to injury prevention in football, coaches, athletics trainers and medical personnel should strive to help educate the player in proper tackling techniques so that these injuries can be minimized. Neck strengthening exercises are important in preventing rapid acceleration/deceleration injuries that can occur without a direct blow to the head. In addition, proper equipment and maintenance, including adequate hel-

met fit (inflation of air bladder in helmet) and shock-absorbing mouthpieces, are essential in preventing concussions. All medical personnel need to be reminded that all unconscious student-ath-

letes should be suspected of a cervical-spine injury until proven otherwise. Special care to the cervical spine should always be used in transporting an unconscious player.

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BAILES EXHIBIT 3

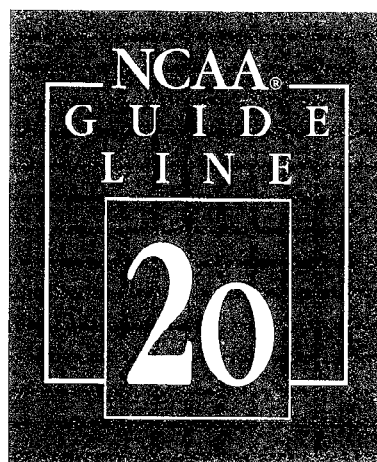
1997-98 NCAA[®] Sports Medicine Handbook



Concussion and Second-Impact Syndrome

June 1994

Revised July 1997



Concussion and the resulting potential complications, such as second-impact syndrome, are potentially life-threatening situations that student-athletes may suffer as a result of their athletics participation. While concussions may occur in almost any contact activity, data from the NCAA Injury Surveillance System (ISS) for the period 1994-96 estimated that more than 1,500 concussions

occur annually in college football. Nine of every 10 head injuries in the sport are reported as concussions. Since no head injury should be considered trivial, proper evaluation and sound decision-making are imperative before the sports medicine profession permits the student-athlete to return to activity.

The definition of concussion is a post-traumatic impairment of

neural status. While loss of consciousness and amnesia have been viewed as the primary components of this injury and have formed the basis for most grading scales, some of the mild concussions, the so-called "bell rung" or "ding," with no resulting loss of consciousness or post-traumatic amnesia, may go unrecognized by coaches, athletic trainers, fellow players or team physicians. The symptoms of concussion (Table 1) vary, depending on the degree and extent of injury. **A student-athlete rendered unconscious for any period of time should not be permitted to return to the practice or game in which the head injury occurred. In addition, no student-athlete should be allowed to return to athletics activity while symptomatic.** Prolonged unconsciousness and neurologic abnormalities suggesting intracranial pathology may require urgent neurosurgical consultation or transfer to a trauma center. If there are any questions as to the severity of past head trauma, or if the trauma required intracranial surgery, clearance of the student-athlete should be deferred until further records are obtained or neurosurgical evaluation is performed.

Several grading scales have been proposed to characterize the degrees, potential severity and return-to-play criteria of





Guideline 2-0 Continued

Concussion and Second-Impact Syndrome

concussion.^{6,7,9,12,13,15} Unfortunately, these categorizations vary and are not universally accepted. Based on the current lack of consensus among the medical community on management of concussions, the NCAA does not endorse any specific concussion grading scale or return-to-play criteria. Although the grading scales and return-to-play criteria currently in the literature may assist in the clinical decision-making for the student-athlete who has suffered a concussion, these grading scales and return-to-play criteria should not be substituted for the clinical judgment of the examining physician.

Post-Concussion Syndrome

After a head injury, the student-athlete may report multiple symptoms (Table 1). While these symptoms usually are short-lived and resolve spontaneously, some individuals may have persistent symptoms after a concussion. Characteristics of post-concussion syndrome are symptoms such as impaired memory and concentration, persistent headache, fatigue, mood and sleep disturbances and dizziness. The student-athlete with symptoms of post-concussion syndrome should not be considered for return to physical activity until resolution of symptoms occurs. Diagnostic studies such as

MRI or CT imaging and/or neuropsychological testing may be indicated and referral to a neurologist or neurosurgeon should be considered.

Multiple Concussions

The athlete who suffers one concussion may be at greater risk for another. Evidence of cognitive impairment and neuroanatomical damage has been reported in some individuals. The number and degree of concussions necessary for permanent impairment is unknown. Return-to-play decisions should be made on an individual basis after the student-athlete has full recovery of neuronal function and can be informed of the potential risks for subsequent concussion and possible complications. As with all concussions, careful review of the

mechanism of injury and appropriate changes in the environment that can be made to reduce the likelihood of subsequent concussion should be undertaken.

Second-Impact Syndrome

The medical staff needs to be aware of the rare but often fatal consequence of the second-impact syndrome. This occurs when an individual sustains a second, often minor trauma to the head before the initial symptoms of the first head injury have resolved. The resulting loss of autoregulation of the brain's blood supply could result in vascular engorgement and herniation of the lower brain, causing death. There is a high mortality rate associated with second-impact syndrome.

Table 1
Symptoms of Concussion

Headache	Irritability
Confusion/Disorientation	Hyperexcitability
Tinnitus	Loss of Consciousness
Dizziness	Unsteadiness
Nausea	Visual Disturbance
Amnesia	Concentration Difficulty
Post-traumatic	
Retrograde	



Guideline 2-0 Continued

Concussion and Second-Impact Syndrome

Summary

The attending medical staff should not allow a player to resume participation in physical activity while the injured student-athlete is recovering from his/her post-concussive symptoms. All individuals involved in sports, including coaches, athletic trainers, team physicians, student-athletes and parents should be educated in the symptoms of concussion and the need for medical attention in the

event of such an injury. With regard to injury prevention in football, coaches, athletic trainers and medical personnel should strive to help educate players in proper tackling techniques so that these injuries can be minimized. Neck-strengthening exercises are important in preventing rapid acceleration/deceleration injuries that can occur without a direct blow to the head. In addition, proper equipment and maintenance, including adequate helmet

fit (inflation of air bladder in helmet) and shock-absorbing mouthpieces, can be beneficial in preventing concussions. All medical personnel need to be reminded that they should suspect all unconscious student-athletes to have suffered a cervical spine injury until proven otherwise. Special care to the cervical spine should always be used in transporting an unconscious player.

References

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Guideline 2-0 Continued

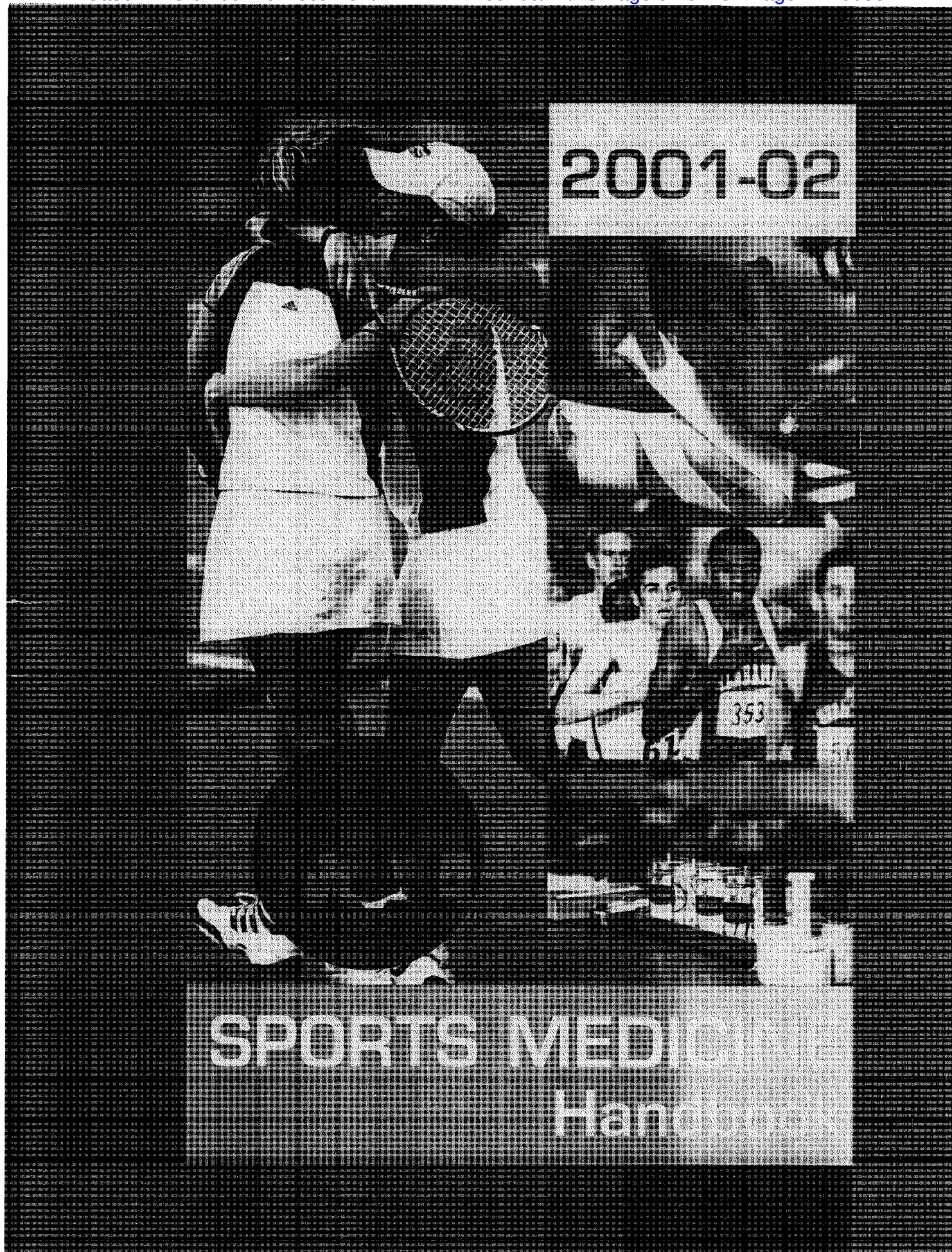
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BAILES EXHIBIT 4



NCAA®

GUIDELINE 20

Concussion and Second-Impact Syndrome

June 1994 • Revised July 1997

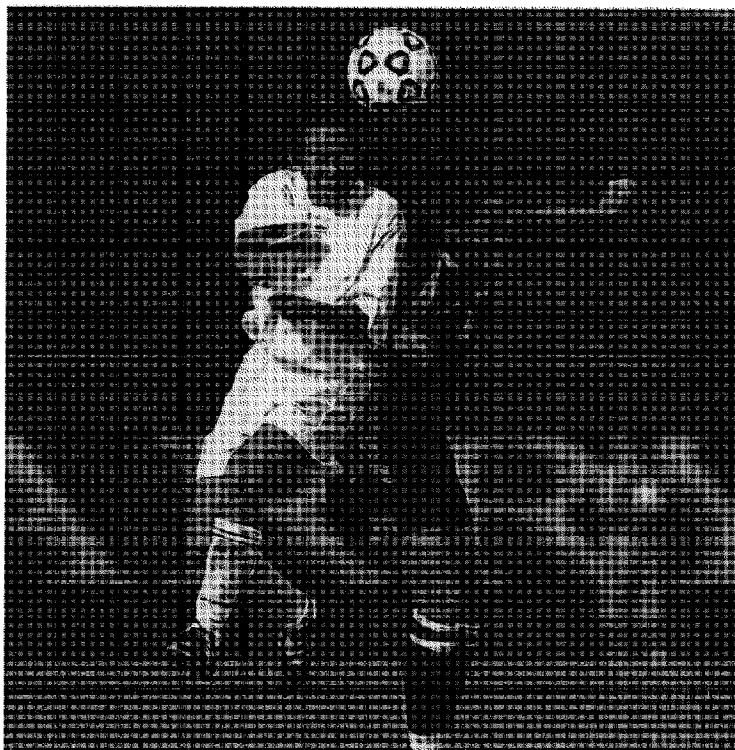
Concussion and the resulting potential complications, such as second-impact syndrome, are potentially life-threatening situations that student-athletes may suffer as a result of their athletics participation. While concussions may occur in almost any contact activity, data from the NCAA Injury Surveillance System (ISS) for the period 1994-96 estimated that more than 1,500 concussions occur annually in college

football. Nine of every 10 head injuries in the sport are reported as concussions. Since no head injury should be considered trivial, proper evaluation and sound decision-making are imperative before the sports medicine profession permits the student-athlete to return to activity.

The definition of concussion is a post-traumatic impairment of neural status. While loss of con-

sciousness and amnesia have been viewed as the primary components of this injury and have formed the basis for most grading scales, some of the mild concussions, the so-called "bell rung" or "ding," with no resulting loss of consciousness or post-traumatic amnesia, may go unrecognized by coaches, athletic trainers, fellow players or team physicians. The symptoms of concussion (Table 1) vary, depending on the degree and extent of injury. **A student-athlete rendered unconscious for any period of time should not be permitted to return to the practice or game in which the head injury occurred. In addition, no student-athlete should be allowed to return to athletics activity while symptomatic.** Prolonged unconsciousness and neurologic abnormalities suggesting intracranial pathology may require urgent neurosurgical consultation or transfer to a trauma center. If there are any questions as to the severity of past head trauma, or if the trauma required intracranial surgery, clearance of the student-athlete should be deferred until further records are obtained or neurosurgical evaluation is performed.

Several grading scales have been proposed to characterize the degrees, potential severity and return-to-play criteria of



Concussion and Second-Impact Syndrome

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After a head injury, the student-athlete may report multiple symptoms (Table 1). While these symptoms usually are short-lived and resolve spontaneously, some individuals may have persistent symptoms after a concussion. Characteristics of post-concussion syndrome are symptoms such as impaired memory and concentration, persistent headache, fatigue, mood and sleep disturbances and dizziness. The student-athlete with symptoms of post-concussion syndrome should not be considered for return to physical activity until resolution of symptoms occurs. Diagnostic studies such as MRI or CT imaging and/or neu-

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Multiple Concussions

The athlete who suffers one concussion may be at greater risk for another. Evidence of cognitive impairment and neuroanatomical damage has been reported in some individuals. The number and degree of concussions necessary for permanent impairment is unknown. Return-to-play decisions should be made on an individual basis after the student-athlete has full recovery of neuronal function and can be informed of the potential risks for subsequent concussion and possible complications. As with all concussions, careful review of the mechanism of injury and appropri-

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The medical staff needs to be aware of the rare but often fatal consequence of the second-impact syndrome. This occurs when an individual sustains a second, often minor trauma to the head before the initial symptoms of the first head injury have resolved. The resulting loss of autoregulation of the brain's blood supply could result in vascular engorgement and herniation of the lower brain, causing death. There is a high mortality rate associated with second-impact syndrome.

Table 1
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Headache	Irritability
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Dizziness	Unsteadiness
Nausea	Visual Disturbance
Amnesia	Concentration Difficulty
Post-traumatic Retrograde	

Concussion and Second-Impact Syndrome

Summary

The attending medical staff should not allow a player to resume participation in physical activity while the injured student-athlete is recovering from his/her post-concussive symptoms. All individuals involved in sports, including coaches, athletic trainers, team physicians, student-athletes and parents should be educated in the symptoms of concussion and the need for medical attention in the

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References

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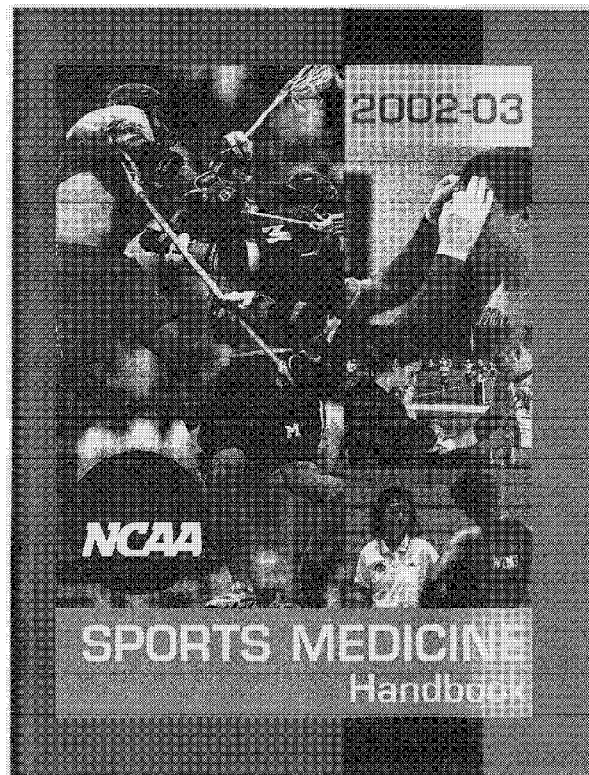


3



**SPECIAL
POPULATIONS**

BAILES EXHIBIT 5



NCAA®

GUIDELINE 20

Concussion and Second-Impact Syndrome

June 1994 • Revised June 2002

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The definition of concussion is a post-traumatic impairment of neural status. While loss of consciousness and amnesia have been viewed as the primary components

of this injury and have formed the basis for most grading scales, some of the mild concussions, the so-called "bell rung" or "ding," with no resulting loss of consciousness or post-traumatic amnesia, may go unrecognized by coaches, athletic trainers, fellow players or team physicians. The symptoms of concussion (Table 1) vary, depending on the degree and extent of injury. **A student-athlete rendered unconscious for any period of time should not be permitted to return to the practice or game in which the head injury occurred. In addition, no student-athlete should be allowed to return to athletics activity while symptomatic.** Prolonged unconsciousness and neurologic abnormalities suggesting intracranial pathology may require urgent neurosurgical consultation or transfer to a trauma center. If there are any questions as to the severity of past head trauma, or if the trauma required intracranial surgery, clearance of the student-athlete should be deferred until further records are obtained or neurosurgical evaluation is performed.

Several grading scales have been proposed to characterize the degrees, potential severity and return-to-play criteria of concussion. Unfortunately, these categorizations vary and are not universally accepted. Based on the



Concussion and Second-Impact Syndrome

current lack of consensus among the medical community on management of concussions, the NCAA does not endorse any specific concussion grading scale or return-to-play criteria. Although the grading scales and return-to-play criteria currently in the literature may assist in the clinical decision-making for the student-athlete who has suffered a concussion, these grading scales and return-to-play criteria should not be substituted for the clinical judgment of the examining physician.

Post-Concussion Syndrome

After a head injury, the student-athlete may report multiple symptoms (Table 1). While these symptoms usually are short-lived and resolve spontaneously, some individuals may have persistent symptoms after a concussion. Characteristics of post-concussion syndrome are symptoms such as impaired memory and concentration, persistent headache, fatigue, mood and sleep disturbances and dizziness. The student-athlete with symptoms of post-concussion syndrome should not be considered for return to physical activity until resolution of symptoms occurs. Diagnostic studies such as MRI or CT imaging and/or neuropsychological testing may be indicated and referral to a neurologist or neurosurgeon should be considered.

Multiple Concussions

The student-athlete who suffers one concussion may be at greater risk for another. Evidence of cognitive impairment and neuroanatomical damage has been reported in some individuals. The number and degree of concussions necessary for permanent impairment is unknown. Return-to-play decisions should be made on an individual basis after the student-athlete has full recovery of neuronal function and can be informed of the potential risks for subsequent concussion and possible complications. As with all concussions, careful review of the mechanism of injury and appropriate changes in the environment that can be made to reduce the likelihood of subsequent concussion should be undertaken.

Second-Impact Syndrome

The medical staff needs to be aware of the rare but often fatal consequence of the second-impact syndrome. This occurs when an individual sustains a second, often minor trauma to the head before the initial symptoms of the first head injury have resolved. The resulting loss of autoregulation of the brain's blood supply could result in vascular engorgement and herniation of the lower brain, causing death. There is a high mortality rate associated with second-impact syndrome.

Summary

The attending medical staff should not allow a player to resume participation in physical activity while the injured student-athlete is recovering from his/her post-concussive

Table 1
Symptoms of Concussion

Headache	Irritability
Confusion/Disorientation	Hyperexcitability
Tinnitus	Loss of Consciousness
Dizziness	Unsteadiness
Nausea	Visual Disturbance
Amnesia	Concentration Difficulty
Post-traumatic	
Retrograde	

Concussion and Second-Impact Syndrome

symptoms. All individuals involved in sports, including coaches, athletic trainers, team physicians, student-athletes and parents, should be educated in the symptoms of concussion and the need for medical attention in the event of such an injury. With regard to injury prevention in football, coaches, athletic trainers and medical personnel should strive to help educate play-

ers in proper tackling techniques so that these injuries can be minimized. Neck-strengthening exercises are important in preventing rapid acceleration/deceleration injuries that can occur without a direct blow to the head. In addition, proper equipment and maintenance, including adequate helmet fit (inflation of air bladder in helmet) and shock-absorbing mouth-

pieces, can be beneficial in preventing concussions. All medical personnel need to be reminded that they should suspect all unconscious student-athletes to have suffered a cervical spine injury until proven otherwise. Special care to the cervical spine should always be used in transporting an unconscious player.

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BAILES EXHIBIT 6



NCAA®

GUIDELINE 2i

Concussion or Mild Traumatic Brain Injury (mTBI) in the Athlete

June 1994 • Revised July 2004

Over 300,000 concussions occur every year, and participation in sport is a common cause of these injuries. These injuries are often difficult to detect, with athletes often underreporting their injury, minimizing their importance, or not recognizing that an injury has occurred. At the college level, these injuries are more common in certain sports such as football, ice hockey, men's and women's soccer, and men's lacrosse. However, they also account for a significant percentage of injuries in men's and women's basketball, women's lacrosse, and other sports traditionally considered "non-contact".

The incidence in helmeted versus non-helmeted sports is also similar. In the years 2000-2002, the rate of concussion during games per 1000 athlete exposures for football was 3.1, for men's ice hockey 2.4, for men's wrestling 1.6 and for men's lacrosse 1.4, respectively, 2.4 for women's ice hockey, 2.1 for women's soccer, 1.7 for men's soccer, 0.8 for field hockey, 0.8 for women's lacrosse, 0.7 for women's basketball, and 0.5 for men's basketball, accounting for between 6.4 and 18.3% of the injuries for these sports as reported by the NCAA Injury Surveillance System (ISS).

Assessment and management of concussive injuries, and return to play decisions remain some of the most difficult responsibilities facing the sports medicine team. There are potentially serious complications of multiple or severe concussions including second impact syndrome, post-concussive syndrome, or post-traumatic encephalopathy. Though there is some controversy as to the existence of second impact syndrome, where a second impact with potentially catastrophic consequences occurs prior to the full recovery after a first insult, the risks include severe cognitive compromise as well as death. Other associated injuries which can occur in the setting of concussion include seizures, cervical spine injuries, skull fractures, and/or intracranial bleed. Due to the serious nature of mild traumatic brain injury, as well as these serious potential complications, it is imperative that the health care professionals taking care of athletes are able to recognize, evaluate, and treat these injuries in a complete and progressive fashion.

Concussion or mild traumatic brain injury (mTBI) has been defined as "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces." Although concussion most commonly occurs after a direct blow to the head, it can occur after a blow elsewhere that is transmitted



Concussion or Mild Traumatic Brain Injury

to the head. Concussions can be defined by the clinical features, pathophysiological changes and / or biomechanical forces that occur, and these have been described in the literature. The neurochemical and neurometabolic changes that occur in concussive injury have been elucidated, and exciting research is underway describing the genetic factors that may play a role in determining which individuals are at an increased risk for sustaining brain injury.

Most commonly, concussion is characterized by the rapid onset of cognitive impairment that is self limited and spontaneously resolves. The acute symptoms of concussion, listed below, are felt to reflect a functional disturbance in cognitive function instead of structural abnormalities, which is why diagnostic tests such as magnetic resonance imaging (MRI) and computerized tomography (CT) scans are most often normal. These studies may have their role in assessing and evaluating the head injured athlete whenever there is concern for the associated injuries of skull fracture, intracranial bleed, seizures, when there is concern for structural abnormalities or when the symptoms of an athlete persist or deteriorate.

Concussion is associated with clinical scenarios that often clear spontaneously, and may or may not be associated with loss of con-

sciousness (LOC).

The sideline evaluation of the brain injured athlete should include an assessment of airway, breathing, and circulation (ABC's), followed by an assessment of the cervical spine and skull for associated injury. The sideline evaluation should also include a neurological and mental status examination and some form of brief neurocognitive testing to assess memory function and attention. This can be in the form of questions regarding the particular practice or competition, previous game results, and remote and recent memory, as well as questions to test the athlete's recall of words, months of the year backwards and calculations. Special note should be made regarding the presence and duration of retrograde or anterograde amnesia, as well as the presence and duration of confusion. A timeline of injury and the

presence of symptoms should be noted. These sideline tests should be performed and repeated as necessary, but do not take the place of other comprehensive neuropsychological tests.

Once an injury occurs and an initial assessment has been made, it is important to determine an initial plan of action, which includes deciding on whether additional referral to a physician and/or emergency department should take place, as well as determining the follow-up care. The medical staff should also determine whether additional observation or hospital admission should be considered.

Follow up care and instructions should be given to the athlete, and ensuring that they are not left alone for an initial period of time should be considered. Athletes should avoid alcohol or other substances

Table 1
SIGNS AND SYMPTOMS OF mTBI

Loss of consciousness (LOC)	Visual Disturbances
Confusion	(Photophobia, blurry vision, photophobia vision, double vision)
Post-traumatic amnesia (PTA)	
Retrograde amnesia (RGA)	
Disorientation	Disequilibrium
Delayed verbal and motor responses	Feeling "in a fog", "zoned out"
Inability to focus	Vacant stare
Headache	Emotional lability
Nausea / Vomiting	Dizziness
Excessive drowsiness	Slurred/ incoherent speech

Concussion or Mild Traumatic Brain Injury

that will impair their cognitive function, and also avoid aspirin and other medications that can increase their risk of bleeding.

As mentioned previously, conventional imaging studies such as MRI and CT scans are usually normal in mTBI. However, these studies are considered an adjunct when any structural lesion, such as an intracranial bleed or fracture, is suspected. If an athlete experiences prolonged loss of consciousness, confusion, seizure activity, focal neurologic deficits, or persistent clinical or cognitive symptoms, then additional testing may be indicated.

There are several grading systems and return to play guidelines in the literature regarding concussion in sport (AAN, Torg, Cantu). However, there may be limitations because they presume that LOC is associated with more severe injuries. It has been demonstrated that LOC does not correlate with severity of injury in patients presenting to an emergency depart-

ment with closed head injury, and has also been demonstrated in athletes with concussion. (Lovell '99). It has been further demonstrated that retrograde amnesia (RGA), post traumatic amnesia (PTA), as well as the duration of confusion & mental status changes greater than 5 minutes may be more sensitive indicators of injury severity (Collins '03). More recent grading systems have been published which attempt to take into account the expanding research in the field of mTBI in athletes. Though it is useful to become familiar with these guidelines, it is important to remember that many of these injuries are best treated in an individual fashion (Cantu '01, Vienna Conference, NATA '04)

Several recent publications have endorsed the use of neurocognitive or neuropsychological testing as the cornerstone of concussion evaluation. These tests provide a reliable assessment and quantification of brain function by examining brain-behavior relationships. These tests are designed to measure a broad

range of cognitive function including speed of information processing, memory recall, attention and concentration, reaction time, scanning and visual tracking ability, and problem solving ability. Several computerized versions of these tests have also been designed to improve the availability of these tests, and make them easier to distribute and utilize. Ideally, these tests are performed prior to the season as a "baseline" with which post-injury tests can be compared. Despite the utility of neuropsychological test batteries in the assessment and treatment of concussion in athletes, several questions remain unanswered. Further research is needed to understand the complete role of neuropsychological testing.

Given these limitations, it is essential that the medical care team taking care of athletes continue to rely on their clinical skills in evaluating the head injured athlete to the best of their ability. It is essential that no athlete be allowed to return to participation when any symptoms, including mild headache, persist. It has also been recommended that for any injury which involves significant symptoms, long duration of symptoms, or difficulties with memory function (either retrograde or anterograde) not be allowed to return to play during the same day of competition. The duration of time that an athlete should be kept

Table 2
SYMPTOMS OF POST-CONCUSSION SYNDROME

Loss of intellectual capacity	Fatigue
Poor recent memory	Irritability
Personality changes	Phono/ photophobia
Headaches	Sleep disturbances
Dizziness	Sleep disturbances
Lack of concentration	Depressed mood
Poor attention	Anxiety

Concussion or Mild Traumatic Brain Injury

out of physical activity is unclear, and in most instances, individualized return to play decisions should be made. These decisions will often depend on the clinical symptoms, as well as previous history of concussion, and severity of previous concussions. Additional factors include the sport, position, age, support system for the athlete, and the overall "readiness" of the athlete to return to sport.

Once an athlete is completely asymptomatic the return to play

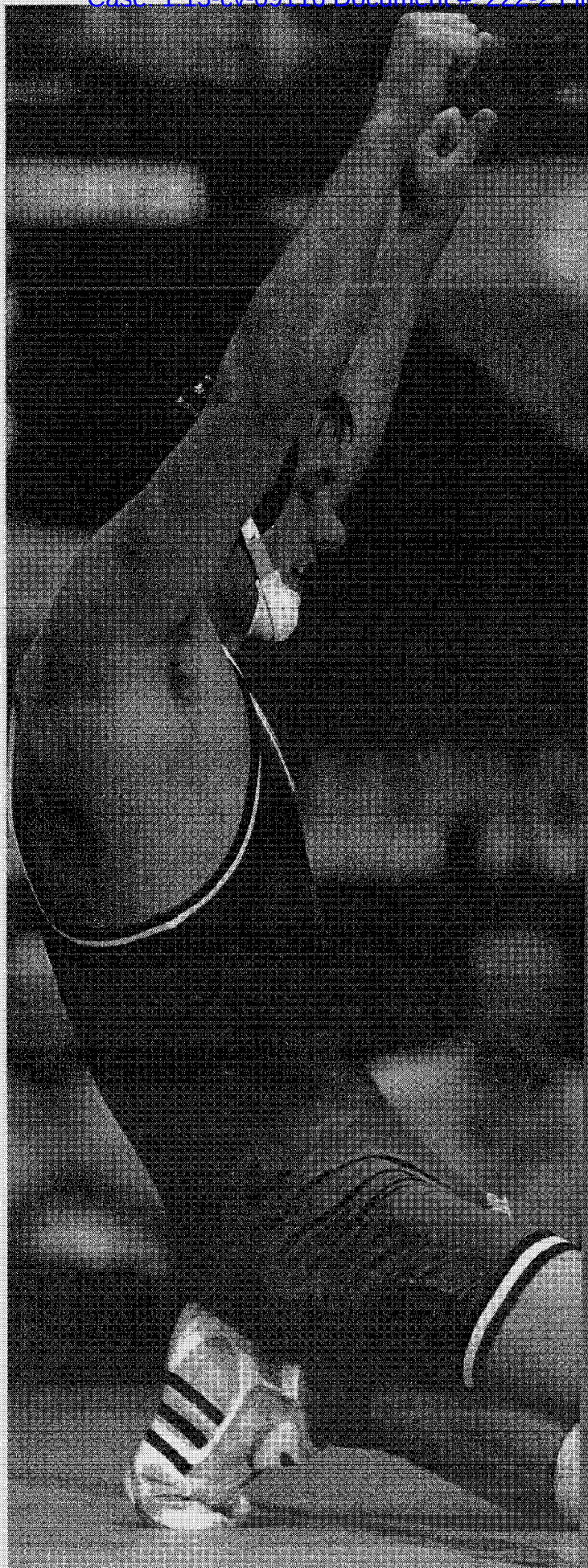
progression should occur in a stepwise fashion with gradual increments in physical exertion and risk of contact. After a period of remaining asymptomatic, the first step is an "exertional challenge" where the athlete exercises for 15-20 minutes in an activity such as biking or running where they increase their heartrate and break a sweat. If they do not experience any symptoms, this can be followed by a steady increase in exertion, followed by return to sport-specific activities that

do not put the athlete at risk for contact. Examples include dribbling a ball or shooting, stickwork or passing, or other agilities. This allows the athlete to return to the practice setting albeit in a limited role. Finally, the athlete can be progressed to practice activities with limited then full contact and finally full contact. How quickly one moves through this progression remains controversial.

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BAILES EXHIBIT 7



2009-10 NCAA®
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Handbook

NCAA® GUIDELINE 2i

Concussion or Mild Traumatic Brain Injury (mTBI) in the Athlete

June 1994 • Revised July 2004

More than 300,000 concussions occur every year, and participation in sport is a common cause of these injuries. These injuries are often difficult to detect, with athletes often underreporting their injury, minimizing their importance or not recognizing that an injury has occurred. At the college level, these injuries are more common in certain sports, such as football, ice hockey, men's and women's soccer, and men's lacrosse. However, they also account for a significant percentage of injuries in men's and women's basketball, women's lacrosse, and other sports

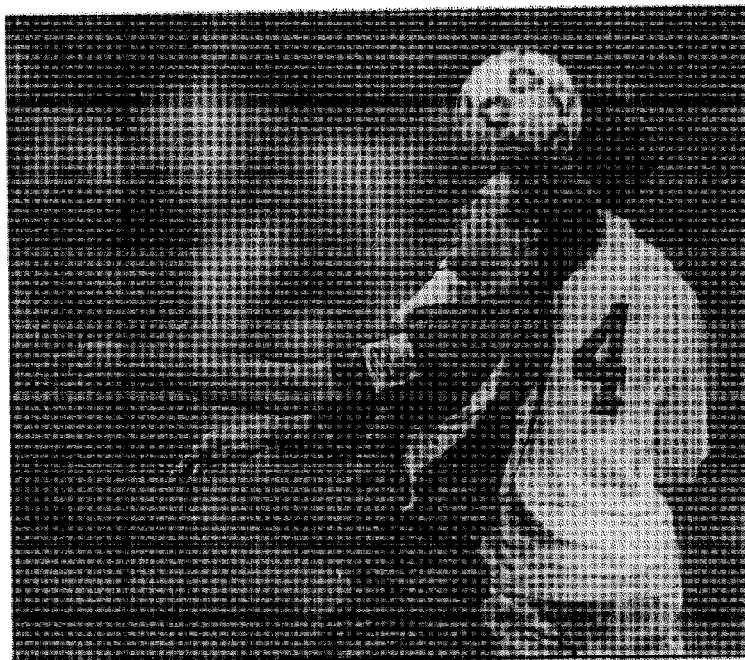
traditionally considered "noncontact."

The incidence in helmeted versus nonhelmeted sports is also similar. In the years 2000 to 2002, the rate of concussion during games per 1,000 athlete exposures for football was 3.1, for men's ice hockey 2.4, for men's wrestling 1.6, for men's lacrosse 1.4, for women's ice hockey 2.4, for women's soccer 2.1, for men's soccer 1.7, for field hockey 0.8, for women's lacrosse 0.8, for women's basketball 0.7, and for men's basketball 0.5, accounting for between 6.4 and 18.3 percent of the injuries for

these sports as reported by the NCAA Injury Surveillance System (ISS).

Assessment and management of concussive injuries, and return-to-play decisions remain some of the most difficult responsibilities facing the sports medicine team. There are potentially serious complications of multiple or severe concussions, including second impact syndrome, postconcussive syndrome, or post-traumatic encephalopathy. Though there is some controversy as to the existence of second impact syndrome, in which a second impact with potentially catastrophic consequences occurs before the full recovery after a first insult, the risks include severe cognitive compromise and death. Other associated injuries which can occur in the setting of concussion include seizures, cervical spine injuries, skull fractures and/or intracranial bleed. Due to the serious nature of mild traumatic brain injury, and these serious potential complications, it is imperative that the health care professionals taking care of athletes are able to recognize, evaluate and treat these injuries in a complete and progressive fashion.

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Concussion or Mild Traumatic Brain Injury

process affecting the brain, induced by traumatic biomechanical forces." Although concussion most commonly occurs after a direct blow to the head, it can occur after a blow elsewhere that is transmitted to the head. Concussions can be defined by the clinical features, pathophysiological changes and / or biomechanical forces that occur, and these have been described in the literature. The neurochemical and neurometabolic changes that occur in concussive injury have been elucidated, and exciting research is underway describing the genetic factors that may play a role in determining which individuals are at an increased risk for sustaining brain injury.

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The sideline evaluation of the brain-injured athlete should include an assessment of airway, breathing, and circulation (ABC's), followed by an assessment of the cervical spine and skull for associated injury. The sideline evaluation should also include a neurological and mental status examination and some form of brief neurocognitive testing to assess memory function and attention. This can be in the form of questions regarding the particular practice or competition, previous game results, and remote and recent memory, and questions

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Once an injury occurs and an initial assessment has been made, it is important to determine an initial plan of action, which includes deciding on whether additional referral to a physician and/or emergency department should take place, and determining the follow-up care. The medical staff should also determine whether additional

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Nausea/Vomiting	Dizziness
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Concussion or Mild Traumatic Brain Injury

observation or hospital admission should be considered.

Follow-up care and instructions should be given to the athlete, and ensuring that they are not left alone for an initial period of time should be considered. Athletes should avoid alcohol or other substances that will impair their cognitive function, and also avoid aspirin and other medications that can increase their risk of bleeding.

As mentioned previously, conventional imaging studies such as MRI and CT scans are usually normal in mTBI. However, these studies are considered an adjunct when any structural lesion, such as an intracranial bleed or fracture, is suspected. If an athlete experiences prolonged loss of consciousness, confusion, seizure activity, focal neurologic deficits or persistent clinical or cognitive symptoms, then additional testing may be indicated.

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Table 2
SYMPTOMS OF POST-CONCUSSION SYNDROME

Loss of intellectual capacity	Fatigue
Poor recent memory	Irritability
Personality changes	Phono/photophobia
Headaches	Sleep disturbances
Dizziness	Sleep disturbances
Lack of concentration	Depressed mood
Poor attention	Anxiety

Concussion or Mild Traumatic Brain Injury

Once an athlete is completely asymptomatic, the return-to-play progression should occur in a step-wise fashion with gradual increments in physical exertion and risk of contact. After a period of remaining asymptomatic, the first step is an "exertional challenge" in which the athlete exercises for 15 to 20 minutes in an activity such as biking or running in which he/she increases his/her heart rate and breaks a sweat. If he/she does not experience any symptoms, this can be followed by a steady increase in exertion, followed by return-to-sport-specific activities that do not put the athlete at risk for contact. Examples include dribbling a ball

1. Heads Up: Concussion Tool Kit

CDC. Available at www.cdc.gov/ncipc/tbi/coaches_tool_kit.htm.

2. Heads Up Video

NATA. Streaming online at www.nata.org/consumer/headsup.htm.

or shooting, stickwork or passing, or other activities. This allows the athlete to return to the practice setting, albeit in a limited role. Finally, the athlete can be progressed to practice activities with limited contact and finally full contact. How quickly one moves through this progression remains controversial.

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BAILES EXHIBIT 8



2010-11 NCAA[®] **Sports Medicine Handbook**



NCAA® GUIDELINE 2i

Concussion or Mild Traumatic Brain Injury (mTBI) in the Athlete

June 1994 • Revised July 2004, 2009, July 2010

Estimates suggest that 1.6-1.8 million concussions occur from participation in sports- and recreation-related activities every year (see reference No. 18). These injuries are often difficult to detect, with athletes often underreporting their injury, minimizing their importance or not recognizing that an injury has occurred. At the college level, these injuries are more common in certain sports, such as football, ice hockey, men's and women's soccer, and men's lacrosse. However, they also

account for a significant percentage of injuries in men's and women's basketball, women's lacrosse, and other sports traditionally considered "noncontact."

The incidence in helmeted versus nonhelmeted sports is also similar. In the years 2004 to 2009, the rate of concussion during games per 1,000 athlete exposures for football was 3.1, for men's lacrosse 2.6, for men's ice hockey 2.4, for women's ice hockey 2.2, for women's soccer 2.2, for wrestling 1.4, for men's

soccer 1.4, for women's lacrosse 1.2, for field hockey 1.2, for women's basketball 1.2, and for men's basketball 0.6, accounting for between 4 and 16.2 percent of the injuries for these sports as reported by the NCAA Injury Surveillance Program by the Datalys Center.

Assessment and management of concussive injuries, and return-to-play decisions remain some of the most difficult responsibilities facing the sports medicine team. There are potentially serious complications of multiple or severe concussions, including second impact syndrome, postconcussive syndrome, or post-traumatic encephalopathy. Though there is some controversy as to the existence of second impact syndrome, in which a second impact with potentially catastrophic consequences occurs before the full recovery after a first insult, the risks include severe cognitive compromise and death. Other associated injuries that can occur in the setting of concussion include seizures, cervical spine injuries, skull fractures and/or intracranial bleed. Due to the serious nature of mild traumatic brain injury, and these serious potential complications, it is



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imperative that the health care professionals taking care of athletes are able to recognize, evaluate and treat these injuries in a complete and progressive fashion. In April 2010, the NCAA Executive Committee adopted a policy that requires NCAA institutions to have a concussion management plan on file. (See information box on page 55.)

Concussion or mild traumatic brain injury (mTBI) has been defined as “a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces.” Although concussion most commonly occurs after a direct blow to the head, it can occur after a blow elsewhere that is transmitted to the head. Concussions can be defined by the clinical features, pathophysiological changes and/or biomechanical forces that occur, and these have been described in the literature. The neurochemical and neurometabolic changes that occur in concussive injury have been elucidated, and exciting research is underway describing the genetic factors that may play a role in determining which individuals are at an increased risk for sustaining brain injury.

Most commonly, concussion is characterized by the rapid onset of cognitive impairment that is self limited and spontaneously resolves. The acute symptoms of concussion, listed below, are felt to reflect a functional disturbance in cognitive function instead of structural abnormalities, which is why diagnostic tests such as magnetic resonance imaging (MRI) and computerized tomography (CT) scans are most often normal. These studies may have their role in assessing and evaluating the head-injured athlete whenever there is concern for the associated

injuries of skull fracture, intracranial bleeding and seizures, when there is concern for structural abnormalities or when the symptoms of an athlete persist or deteriorate.

Concussion is associated with clinical scenarios that often clear spontaneously, and may or may not be associated with loss of consciousness (LOC).

The sideline evaluation of the brain-injured athlete should include an assessment of airway, breathing and circulation (ABCs), followed by an assessment of the cervical spine and skull for associated injury. The sideline evaluation should also include a neurological and mental status examination and some form of brief neurocognitive testing to assess memory function and attention. This can be in the form of questions regarding the particular practice or competition, previous game results, and remote and recent memory, and questions to test the athlete’s recall of words, months of the year backwards and calculations. Special note should be made regarding the presence and duration of retrograde or anterograde amnesia, and the presence and duration of confusion. A timeline of injury and the presence of symptoms should be

noted. These sideline tests should be performed and repeated as necessary, but do not take the place of other comprehensive neuropsychological tests.

Once an injury occurs and an initial assessment has been made, it is important to determine an initial plan of action, which includes deciding on whether additional referral to a physician and/or emergency department should take place, and determining the follow-up care. The medical staff should also determine whether additional observation or hospital admission should be considered.

Follow-up care and instructions should be given to the athlete, and ensuring that they are not left alone for an initial period of time should be considered. Athletes should avoid alcohol or other substances that will impair their cognitive function, and also avoid aspirin and other medications that can increase their risk of bleeding.

As mentioned previously, conventional imaging studies such as MRI and CT scans are usually normal in mTBI. However, these studies are considered an adjunct when any structural lesion, such as an intracranial bleed or fracture, is suspected. If an athlete

Table 1
SIGNS AND SYMPTOMS OF mTBI

Loss of consciousness (LOC)	Visual Disturbances
Confusion	(Photophobia, blurry vision, photophobia vision, double vision)
Post-traumatic amnesia (PTA)	
Retrograde amnesia (RGA)	
Disorientation	Disequilibrium
Delayed verbal and motor responses	Feeling “in a fog,” “zoned out”
Inability to focus	Vacant stare
Headache	Emotional lability
Nausea/Vomiting	Dizziness
Excessive drowsiness	Slurred/incoherent speech

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experiences prolonged loss of consciousness, confusion, seizure activity, focal neurologic deficits or persistent clinical or cognitive symptoms, then additional testing may be indicated.

There are several grading systems and return-to-play guidelines in the literature regarding concussion in sport (AAN, Torg, Cantu). However, there may be limitations because they presume that LOC is associated with more severe injuries. It has been demonstrated that LOC does not correlate with severity of injury in patients presenting to an emergency department with closed head injury, and has also been demonstrated in athletes with concussion (Lovell '99). It has been further demonstrated that retrograde amnesia (RGA), post-traumatic amnesia (PTA), and the duration of confusion and mental status changes are more sensitive indicators of injury severity (Collins '03), thus an athlete with these symptoms should not be allowed to return to play during the same day. These athletes should not return to any participation until cleared by a physician. More recent grading systems have been published that attempt to take into account the expanding research in the field of mTBI in athletes. Though it is useful to become familiar with these guidelines, it is important to remember that many of these injuries are best treated in an

1. NCAA Concussion Fact Sheets and Video for Coaches and Student-Athletes

Available at www.NCAA.org/health-safety.

2. Heads Up: Concussion Tool Kit

CDC. Available at www.cdc.gov/ncipc/tbi/coaches_tool_kit.htm.

3. Heads Up Video

NATA. Streaming online at www.nata.org/consumer/headsup.htm.

individual fashion (Cantu '01, Zurich Conference, NATA '04).

Several recent publications have endorsed the use of neurocognitive or neuropsychological testing as the cornerstone of concussion evaluation. These tests provide a reliable assessment and quantification of brain function by examining brain-behavior relationships. These tests are designed to measure a broad range of cognitive function, including speed of information processing, memory recall, attention and concentration, reaction time, scanning and visual tracking ability, and problem solving ability. Several computerized versions of these tests have also been designed to improve the availability of these tests, and make them easier to distribute and use. Ideally, these tests are performed before the season as a "baseline" with which post-injury tests can be compared. Despite the utility of neuropsychological test batteries in the assessment and treatment of concussion in athletes, several questions remain unanswered. Further research is

needed to understand the complete role of neuropsychological testing.

Given these limitations, it is essential that the medical care team treating athletes continue to rely on its clinical skills in evaluating the head-injured athlete to the best of its ability. It is essential that no athlete be allowed to return to participation when any symptoms persist, either at rest or during exertion. Any athlete exhibiting an injury that involves significant symptoms, long duration of symptoms or difficulties with memory function should not be allowed to return to play during the same day of competition. The duration of time that an athlete should be kept out of physical activity is unclear, and in most instances, individualized return-to-play decisions should be made. These decisions will often depend on the clinical symptoms, previous history of concussion and severity of previous concussions. Additional factors include the sport, position, age, support system for the athlete and the overall "readiness" of the athlete to return to sport.

Once an athlete is completely asymptomatic, the return-to-play progression should occur in a step-wise fashion with gradual increments in physical exertion and risk of contact. After a period of remaining asymptomatic, the first step is an "exertional challenge" in which the athlete exercises for 15 to 20 minutes in an activity such as biking or running in which he/she increases his/her heart rate and

Table 2

SYMPTOMS OF POST-CONCUSSION SYNDROME

Loss of intellectual capacity	Fatigue
Poor recent memory	Irritability
Personality changes	Phono/photophobia
Headaches	Sleep disturbances
Dizziness	Sleep disturbances
Lack of concentration	Depressed mood
Poor attention	Anxiety

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breaks a sweat. If he/she does not experience any symptoms, this can be followed by a steady increase in exertion, followed by return-to-sport-specific activities that do not put the athlete at risk for contact. Examples include dribbling a ball or shooting, stickwork or passing, or other agilities. This allows the athlete to return to the practice setting, albeit in a limited role. Finally, the athlete can be progressed to practice activities with limited contact and finally full contact. How quickly one moves through this progression remains controversial.

The NCAA Executive Committee adopted (April 2010) the following policy for institutions in all three divisions.

"Institutions shall have a concussion management plan on file such that a student-athlete who exhibits signs, symptoms or behaviors consistent with a concussion shall be removed from practice or competition and evaluated by an athletics healthcare provider with experience in the evaluation and management of concussions. Student-athletes diagnosed with a concussion shall not return to activity for the remainder of that day. Medical clearance shall be determined by the team physician or his or her designee according to the concussion management plan.

"In addition, student-athletes must sign a statement in which they accept the responsibility for reporting their injuries and illnesses to the institutional medical staff, including signs and symptoms of concussions. During the review and signing process, student-athletes should be presented with educational material on concussions."

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In Addition to the Executive Committee Policy Requirements, Additional Best Practices for a Concussion Management Plan Include, but are not Limited to:

1. Although sports currently have rules in place, athletics staff, student-athletes and officials should continue to emphasize that purposeful or flagrant head or neck contact in any sport should not be permitted and current rules of play should be strictly enforced.
2. Institutions should have on file and annually update an emergency action plan for each athletics venue to respond to student-athlete catastrophic injuries and illnesses, including but not limited to, concussions, heat illness, spine injury, cardiac arrest, respiratory distress (e.g., asthma), and sickle cell trait collapses. All athletics healthcare providers and coaches should review and practice the plan at least annually.
3. Institutions should have on file an appropriate healthcare plan that includes equitable access to athletics healthcare providers for each NCAA sport.
4. Athletics healthcare providers should be empowered to have the unchallengeable authority to determine management and return-to-play of any ill or injured student-athlete, as the provider deems appropriate. For example, a countable coach should not serve as the primary supervisor for an athletics healthcare provider, nor should the coach have sole hiring or firing authority over a provider.
5. The concussion management plan should outline the roles of athletics healthcare staff (e.g., physician, certified athletic trainer, nurse practitioner, physician assistant, neurologist, neuropsychologist). In addition, the following components have been specifically identified for the collegiate environment:
 - a. Institutions should ensure that coaches have acknowledged that they understand the concussion management plan, their role within the plan and that they received education about concussions.
 - b. Athletics healthcare providers should practice within the standards as established for their professional practice (e.g., physician, certified athletic trainer, nurse practitioner, physician assistant, neurologist, neuropsychologist).
 - c. Institutions should record a baseline assessment for each student-athlete before the first practice in the sports of baseball, basketball, diving, equestrian, field hockey, football, gymnastics, ice hockey, lacrosse, pole vaulting, rugby, soccer, softball, water polo and wrestling, at a minimum. The same baseline assessment tools should be used post-injury at appropriate time intervals. The baseline assessment should consist of one or more of the following areas of assessment.
 - 1) At a minimum, the baseline assessment should consist of the use of a symptoms checklist and standardized cognitive and balance assessments [e.g., SAC; SCAT; SCAT II; Balance Error Scoring System (BESS)].
 - 2) Additionally, neuropsychological testing (e.g., computerized, standard paper and pencil) has been shown to be effective in the evaluation and management of concussions. The development and implementation of a neuropsychological testing program should be performed in consultation with a neuropsychologist who is in the best position to interpret NP tests by virtue of background and training. However, there may be situations in which neuropsychologists are not available and a physician experienced in the use and interpretation of such testing in an athletic population may perform or interpret NP screening tests.
 - d. The student-athlete should receive serial monitoring for deterioration. Athletes should be provided with written instructions upon discharge, preferably with a roommate, guardian or someone who can follow the instructions.
 - e. The student-athlete should be evaluated by a team physician as outlined within the concussion management plan. Once asymptomatic and post-exertion assessments are within normal baseline limits, return-to-play should follow a medically supervised stepwise process.
6. Institutions should document the incident, evaluation, continued management and clearance of the student-athlete with a concussion.

For references, visit www.NCAA.org/health-safety.

EXHIBIT C

**UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION**

)	MDL No. 2492
IN RE NATIONAL COLLEGIATE)	
ATHLETIC ASSOCIATION STUDENT-)	Master Docket No. 1:13-cv-09116
ATHLETE CONCUSSION INJURY)	
LITIGATION)	This Document Relates To:
)	All Cases
)	
)	Judge John Z. Lee
)	
)	Magistrate Judge Geraldine Soat Brown

DECLARATION OF ROSS MISHKIN

I, Ross Mishkin, declare as follows:

1. I am a Managing Director of The Claro Group, LLC (“Claro”). Claro is a consulting firm with wide experience in complex commercial litigation and claims support, including the valuation of claims in class actions as well as mass torts.

2. I earned a B.S. in Finance from The University of Illinois Urbana-Champaign in 1992 and an M.B.A. from The University of Chicago in 2000. During my career, I have been a manager at Arthur Andersen LLP and a principal at LECG, LLC before becoming a Managing Director at Claro.

3. Except as expressly stated to the contrary, I make this declaration based upon personal knowledge. If called as a witness, I would and could testify competently hereto.

4. The National Collegiate Athletic Association (“NCAA”) engaged Claro to provide expert services in the above-captioned litigation. As part of its engagement with the NCAA, Claro developed certain models to estimate, among other things, the adequacy of the

proposed Medical Monitoring Fund.¹ That analysis involved estimating the size of the Settlement Class and the number of Settlement Class Members who sustained concussions while participating in an NCAA athletic event through the 2014-2015 academic year. In preparing these estimates, I relied upon concussion incidence data for the 2009-2012 academic years obtained from the NCAA Injury Surveillance System (the “Injury Surveillance System”). Data collection for the Injury Surveillance System has been managed by the Datalys Center for Sports Injury Research and Prevention (the “Datalys Center”) since the 2009-2010 academic year. Injury data from the Injury Surveillance System was provided by the Datalys Center and provided to me by counsel for the NCAA.

5. The Injury Surveillance System data provided to me does not contain the names of individuals who sustained concussions or head injuries. Moreover, it does not contain any information from which the identities of individuals who sustained concussions or head injuries could be derived, such as address, date and place of birth, and/or Social Security Number. The Injury Surveillance System data I reviewed is anonymized and includes attributes such as sport, division, player position, gender, type of injury, and body part injured, among other fields.

6. Based on conversations I have had with Datalys Center employees who oversee the Injury Surveillance System, it is my understanding that the injury data that the Datalys Center receives from NCAA member institutions does not contain any personally identifiable information. As explained to me, all personally identifiable information is removed by the member institution before the data is exported to the Datalys Center. Underlying injury information about particular student-athletes resides with NCAA member institutions and is

¹ Capitalized terms have the meaning ascribed to them in the Amended Settlement Agreement, which is Exhibit 1 to the joint motion for preliminary approval filed with the Court on April 14, 2015. See Am. Settlement Agt. (Dkt. #154-1).

subject to the various member institutions' document and data retention policies, to the extent such data otherwise still exists.

7. Member institutions provide information to the Injury Surveillance System on a voluntary basis. Not every NCAA team participates in the Injury Surveillance System. I further understand the Injury Surveillance System data is not intended to be an exhaustive database of all head injuries, and I did not independently review or verify the data in the Injury Surveillance System. Not every concussion or head injury sustained by an NCAA student-athlete, however, is reported and thus, not every concussion or head injury sustained by an NCAA student-athlete is reflected in the Injury Surveillance System.

8. The Injury Surveillance System data provided to me does not include information on any subsequent diagnosis a student-athlete may have received. Thus, it does not indicate if the person subsequently was diagnosed with post-concussion syndrome, Parkinson's disease, Alzheimer's disease, chronic traumatic encephalopathy, or any other illness or condition.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge, information and belief.

Executed on: September 12, 2015



Ross Mishkin

EXHIBIT D

EXHIBIT D
DISCOVERY STATUS IN INDIVIDUAL NCAA CONCUSSION-RELATED CASES

Case Name	Non-NCAA Defendants	Number of Document Requests Thus Far ¹	Number of Interrogatories Thus Far	Number of Depositions Thus Far	Damages Pled
<u>Anderson v. NCAA</u> , No. 631093 (La. 19th Dist. Ct. – E. Baton Rouge)	<ol style="list-style-type: none"> 1. Riddell, Inc.² 2. All American Sports Corp. d/b/a Riddell/All American 3. Riddell Sports Group, Inc. 4. Easton-Bell Sports, Inc. 5. Easton-Bell Sports, LLC 6. EB Sports Corp. 7. RBG Holdings Corp. 	0	0	0	No specific amount pled.
<u>Bradley v. NCAA</u> , No. 15-CV-535 (D.D.C.)	<ol style="list-style-type: none"> 1. The Patriot League 2. American University 3. Maryland Sports Medicine Center 4. David L. Higgins, M.D., P.C. 5. David L. Higgins, M.D. 6. Aaron Williams, D.O. 	121	178	0	\$1,000,000 or more

¹ Chart provides number of discovery requests served as of September 14, 2015.

² Of the helmet manufacturers, only Riddell, Inc. remains a party in the case. Compare Petition, Anderson v. NCAA, No. 631093 (La. 19th Dist. Ct. - E. Baton Rouge June 6, 2014), with First Amending Petition, Anderson v. NCAA, No. 631093 (La. 19th Dist. Ct. - E. Baton Rouge Feb. 5, 2015).

Case Name	Non-NCAA Defendants	Number of Document Requests Thus Far ³	Number of Interrogatories Thus Far	Number of Depositions Thus Far	Damages Pled
<u>Calderone v. NCAA</u> , No. 14-706987 (N.Y. Sup. Ct. – Queens Cnty.) ³	<ol style="list-style-type: none"> 1. Molloy College 2. East Coast Conference 3. Daniel Longo 4. Susan Cassidy-Lyke 5. James Zegers 6. Independent Soccer Officials Assigning Bureau, Inc. 7. National Intercollegiate Soccer Officials Association 8. “John Doe” 9. “John Roe” 10. Central Atlantic Collegiate Conference 	0	0	0	\$25,000 or more
<u>Cunningham v. NCAA</u> , No. DC-14-12249 (Tex. 160th Dist. Ct. – Dallas Cnty.)	None	0	0	0	\$1,000,000 or more
<u>Onyshko v. NCAA</u> , No. C-63-CV-201403620 (Pa. Ct. Common Pleas – Washington Cnty.)	None	47	57	17	\$50,000 or more

³ The NCAA was dismissed from this case but was originally named in the Complaint. See Order, Calderone v. NCAA, No. 14-706987 (N.Y. Sup. Ct. – Queens Cnty. July 2, 2015).

Case Name	Non-NCAA Defendants	Number of Document Requests Thus Far ⁴	Number of Interrogatories Thus Far	Number of Depositions Thus Far	Damages Pled
<u>Schmitz v. NCAA</u> , No. CV 14 1834486 (Ohio Ct. Common Pleas – Cuyahoga Cnty.) ⁴	1. University of Notre Dame	0	0	0	\$25,000 or more
<u>Sheely v. NCAA</u> , No. 380569V (Md. Cir. Ct. – Montgomery Cnty.)	1. Jamie Schumacher 2. Thomas Rogish 3. Michael Sweitzer, Jr. 4. Kranos Corp. d/b/a Schutt Sports 5. George L. Heider, Inc. d/b/a Sportman's	506	275	41	\$1,500,000 or more
<u>Walen v. NCAA</u> , No. 14CV12218 (Or. Cir. Ct. – Multnomah Cnty.)	1. Portland State University 2. Oregon Health and Science University 3. Nigel Burton 4. Duane Duey 5. Dr. Charles Webb	213	0	0	No more than \$5,000,000
<u>Wells v. NCAA</u> , No. 02-CV-2013-902657.00 (Ala. Cir. Ct. – Mobile Cnty.)	None	58	33	1	No specific amount pleaded
AVERAGE	3.78	105.33	60.33	6.56	\$1,228,571.43⁵

⁴ The NCAA was dismissed from this case but was originally named in the Complaint. See Order, Schmitz v. NCAA, No. CV 14 1834486 (Ohio Ct. Common Pleas – Cuyahoga Cnty. Sept. 1, 2015).

⁵ Average based on the seven cases in which damages were specifically pled.

EXHIBIT E

EXHIBIT E**CLASS DEFINITIONS IN CONSOLIDATED CASES NOW BEFORE THE COURT**

Case	Class Definition
<u>Arrington v. NCAA</u> (N.D. Ill. No. 1:11-cv-06356)	All persons who are playing or have played an NCAA-sanctioned sport at an NCAA member institution.
<u>Caldwell v. NCAA</u> (N.D. Ill. No. 1:14-cv-00195)	All former NCAA football players residing in the United States, who did not go on to play professional football in the National Football League.
<u>Doughty v. NCAA</u> (N.D. Ill. No. 1:14-cv-00199)	All former NCAA football players residing in the United States, who did not go on to play professional football in the National Football League.
<u>DuRocher v. NCAA</u> (N.D. Ill. No. 1:14-cv-00035)	All former NCAA football players, and spouses of players, who sustained a concussion(s) or suffered concussion-like symptoms while playing football in a NCAA football game, and who have developed or will develop mental or physical problems as a result of the concussion(s) suffered and have incurred or will incur medical expenses from such injuries.
<u>Hudson v. NCAA</u> (N.D. Ill. No. 1:14-cv-00194)	All former NCAA football players residing in the United States, who did not go on to play professional football in the National Football League.
<u>Jackson v. NCAA</u> (N.D. Ill. No. 1:14-cv-04387)	All current and former student-athletes who played NCAA-regulated football in any college or university with membership status in the NCAA, regardless of the time-period or state in which the student-athlete played football.
<u>Morgan v. NCAA</u> (N.D. Ill. No. 1:14-cv-00196)	All former NCAA football players residing in the United States who did not go on to play professional football in the NFL.
<u>Nichols v. NCAA</u> (N.D. Ill. No. 1:14-cv-00962)	All current and former NCAA student-athletes who sustained a concussion(s) or suffered concussion-like symptoms while playing an NCAA-regulated sport and who incurred medical expenses as a result.
<u>Powell v. NCAA</u> (N.D. Ill. No. 1:14-cv-00198)	All former NCAA football players who sustained a concussion(s) or suffered concussion-like symptoms while playing football at an NCAA school, and who have, since ending their NCAA careers, developed chronic headaches, chronic dizziness, sleeplessness or dementia or Alzheimer's disease and/or other physical, cognitive and mental problems as a result of the concussion(s) suffered while a player and who did not play in the NFL.
<u>Walker v. NCAA</u> (N.D. Ill. No. 1:13-cv-09117)	All former NCAA football players residing in the United States, who did not go on to play professional football in the National Football League.

Case	Class Definition
<u>Walton v. NCAA</u> (N.D. Ill. No. 1:14-cv-00200)	All former NCAA football players residing in the United States, who did not go on to play professional football in the National Football League.
<u>Washington v. NCAA</u> (N.D. Ill. No. 1:14-cv-00197)	All former NCAA football players residing in the United States, who did not go on to play professional football in the National Football League.
<u>Whittier v. NCAA</u> (N.D. Ill. No. 1:14-cv-09322)	All former NCAA football players residing in the U.S. who played from 1960 - 2014 who did not go on to play professional football in the NFL and who have been diagnosed with a latent brain injury or disease.
<u>Wolf v. NCAA</u> (N.D. Ill. No. 1:14-cv-01268)	All current and former NCAA student-athletes who played an NCAA sport.

EXHIBIT F
(REDACTED
VERSION,
PUBLICLY FILED)

EXHIBIT F
ISSUES AND DEFENSES LIKELY TO ARISE IN INDIVIDUAL CASES

Issue/Defense	Record Evidence
Assumption of the Risk	<p>Like many members of the Settlement Class, Plaintiff Palacios signed a form indicating she had been told about risks associated with concussions. <u>See</u> Palacios Dep., Ex. 38, at 132:6-19; <u>id.</u> at Ex. 15, PALACIOS-OUACHITA200000942, PALACIOS-OUACHITA200000945.</p> <p>Plaintiff Arrington testified that “there’s assumed risk when you play football” and “I don’t know if they could have did something to prevent it because concussions happen in football.” <u>See</u> Arrington Dep., Ex. 36, at 128:18, 173:9-11.</p> <p>Plaintiff Solomon testified that even before he matriculated to the University of Maine, he knew that playing hockey posed a risk of head injury, but he continued to play because playing hockey was all he had ever wanted to do. <u>See</u> Solomon Dep., Ex. 39, at 47:4-48:18.</p> <p>Plaintiff Owens signed a form acknowledging he had been told of risks of head injuries that might occur while wearing a football helmet during football games or practices. <u>See</u> Owens Dep., Ex. 37, at 286:17-290:3.</p>
Contributory Negligence	<p>Plaintiff Palacios was told to stop playing soccer after sustaining three concussions. On a form she filled out before matriculating to Ouachita Baptist University (“OBU”), she indicated she had suffered three concussions, but she continued to play soccer in college. <u>See</u> Palacios Dep., Ex. 38, at 98:10-99:6.</p> <p>Plaintiff Arrington was told to stop playing football, but he and his parents rejected his doctor’s advice and challenged the school’s refusal to allow him to play. <u>See</u> ARRINGTON000002, Ex. 40.</p> <p>Plaintiff Solomon admitted to lying to his college about his concussion history, because he was afraid that if he admitted to having prior concussions, he would not be able to play hockey. <u>See</u> Solomon Dep., Ex. 39, at 171:11-172:13.</p> <p>Plaintiff Solomon admitted to concealing concussion symptoms so that he could return to play after sustaining a hit to the head, despite knowing that doing so was dangerous. <u>See id.</u> at 84:20-87:9.</p> <p>Plaintiff Owens refused to ever signal a fair catch when fielding punts, although he was aware of the risks and the purpose of the fair catch rule. <u>See</u> Owens Dep., Ex. 37, at 57:4-19, 225:16-</p>

Issue/Defense	Record Evidence
	<p>226:11; NCAA 2012-2013 Rule Book, Ex. 41, at Rule 6 § 5, Art. 1(d) (“The purpose of the fair catch provision is to protect the receiver [of a kick] who, by his fair catch signal, agrees he or a teammate will not advance after the catch.”). It was on such a play that Plaintiff Owens sustained a concussion giving rise to his present claims. <u>See</u> Fourth Am. Compl. (Dkt. #171), at ¶ 39.</p>
Plaintiff’s Knowledge of the Risk of Concussions	<p>Plaintiff Palacios testified at her deposition that she began wearing protective headgear in high school after she “got [her] first serious concussion” playing soccer. <u>See</u> Palacios Dep., Ex. 38, at 23:10-12.</p> <p>After Plaintiff Palacios sustained two concussions during high school, her physician told her that if she sustained a third concussion, she should stop playing soccer. <u>See</u> <i>id.</i> at 33:1-34:1.</p> <p>Plaintiff Solomon testified at his deposition that playing hockey involved a risk of injuries, including head injuries. <u>See</u> Solomon Dep., Ex. 39, at 47:15-48:3.</p> <p>Plaintiff Owens testified at his deposition that he had suffered at least one and possibly two concussions <u>prior</u> to playing football at the University of Central Arkansas. <u>See</u> Owens Dep., Ex. 36, at 36:8-11.</p>
Causes of Plaintiff’s Alleged Injury	<p>Plaintiff Palacios sustained two concussions while playing soccer during high school. <u>See</u> Palacios Dep., Ex. 38, at 27:3-28:17, 29:12-31:21.</p> <p>Plaintiff Solomon testified that while playing junior hockey, prior to his matriculation to the University of Maine, he got hit “very hard” and his head hit the ice “very hard.” <u>See</u> Solomon Dep., Ex. 39, at 33:12-18. He did not recall much after that and had “a very bad headache.” <u>Id.</u> He also testified that while playing junior hockey in 2006, he hit his head on the ice and was knocked unconscious. <u>Id.</u> at 38:20-39:8. He experienced recurring “terrible headaches,” light sensitivity, depression, and felt like he was “in a fog.” <u>Id.</u> at 41:11-21. He was diagnosed with a concussion. <u>Id.</u> at 41:22-42:6.</p> <p>Plaintiff Owens was diagnosed with one concussion and sustained a second possible concussion while playing high school football. <u>See</u> Owens Dep., Ex. 37, at 30:11-32:11.</p> <p style="text-align: center;">REDACTED CONFIDENTIAL</p> <p style="text-align: center;"><u>See</u> Arrington Dep., Ex. 36, at 95:17-96:9.</p> <p style="text-align: center;">REDACTED CONFIDENTIAL</p>

Issue/Defense	Record Evidence
	<p>REDACTED CONFIDENTIAL</p> <p><u>See id.</u> at Ex. 15, ARRINGTON-EIU00000714.</p>
Potentially Liable Third Parties	<p>Plaintiff Owens testified that all but one of the concussions he sustained playing football occurred when he was wearing a helmet. <u>See</u> Owens Dep., Ex. 37, at 42:5-10.</p> <p>Plaintiff Owens testified that he sustained a possible concussion during a voluntary, non-contact college football practice when another player made illegal contact and hit Mr. Owens in the back of the head. <u>Id.</u> at 174:8-176:3.</p> <p>Plaintiff Solomon hit his head and lost consciousness during a hockey game in college. <u>See</u> Solomon Dep., Ex. 39, at 83:11-84:1. He was treated by his team trainer and returned to play in the same game. <u>See id.</u> at 84:5-85:21. When he told the trainer about his concussion symptoms after the game, the trainer told him to go home and rest and did not advise Solomon to see a doctor. <u>See id.</u> at 87:21-89:7.</p> <p>REDACTED CONFIDENTIAL</p> <p><u>See</u> Arrington Dep., Ex. 36, at 74:8-16.</p> <p>REDACTED CONFIDENTIAL</p> <p><u>See id.</u> at 77:21-80:10.</p> <p>Plaintiff Palacios relied on protective headgear to prevent concussions. <u>See</u> Palacios Dep., Ex. 38, at 22:18-23:12.</p> <p>Plaintiff Palacios testified that her coach ordered her to run at soccer practice when she was still experiencing concussion symptoms. <u>See id.</u> at 71:1-11, 73:12-20. His instruction to run while she was symptomatic violated OBU's return-to-play policy. <u>See id.</u> at 139:2-140:14.</p> <p>Plaintiff Palacios testified that after checking with the head athletic trainer by telephone, a student trainer told Palacios she could resume running at soccer practice four days after she sustained a concussion. <u>See id.</u> at 13:5-14:21.</p> <p>Plaintiff Palacios testified that she suffered her only concussion at OBU when another player "threw her head back" and hit Palacios above the eye. <u>See id.</u> at 32:2-15.</p>
Return to Play	<p>Plaintiff Palacios testified that she sustained one concussion in college and she quit playing soccer after that concussion. <u>See</u> Palacios Dep., Ex. 38, at 8:6-9:8, 21:4-8, 39:3-8.</p> <p>Plaintiff Solomon hid concussion symptoms so that he could</p>

Issue/Defense	Record Evidence
	return to play during a college hockey game. <u>See</u> Solomon Dep., Ex. 39, at 83:11-86:21.
Failure to Mitigate Damages	<p>Plaintiff Palacios testified that after sustaining her one and only college concussion, her mother advised her to see a doctor immediately. <u>See</u> Palacios Dep., Ex. 38, at 58:16-59:1. Palacios testified that although she experienced severe concussion symptoms during the days immediately following her concussion, she did not go to a doctor until several days later when she visited a doctor to receive treatment for her eye. She could not recall if she told that doctor she was experiencing concussion symptoms. <u>See id.</u> at 66:7-22, 67:25-68:10, 69:2-70:2.</p> <p>Plaintiff Solomon testified that he sustained a head injury while playing hockey in 2007, before he matriculated to the University of Maine. <u>See</u> Solomon Dep., Ex. 39, at 69:12-23. He testified that after hitting his head, he felt “punchdrunk,” “dizzy,” was “slurring his words” and “saying things that made no sense whatsoever.” <u>See id.</u> at 69:19-23, 70:6-12. He told his parents and coach about his symptoms but did not recall seeking medical attention after this injury even though he suspected he had suffered a concussion. <u>See id.</u> at 70:21-71:3, 72:18-73:4.</p> <p>Plaintiff Owens testified that, as far as he could recall, he did not seek medical care after sustaining a possible concussion during a football game his senior year in high school, despite having a headache and vomiting after the game. <u>See</u> Owens Dep., Ex. 37, at 30:11-19, 31:10-32:11, 47:6-13.</p> <p style="text-align: center;">REDACTED CONFIDENTIAL</p> <p style="text-align: right;"><u>See</u></p> <p>Arrington Dep., Ex. 36, at 98:2-22. Plaintiff Arrington pleaded to be allowed to continue playing football. <u>See id.</u> at 103:1-8, 112:19-113:15, 118:12-19. Ultimately, Arrington did play football his senior year. <u>See id.</u> at 106:5-10.</p>

EXHIBIT G

EXHIBIT G**CASES ON WHICH NICHOLS PRINCIPALLY RELIES TO ARGUE THAT MANAGEABILITY CONCERNS DO NOT PRECLUDE CERTIFICATION OF A PERSONAL INJURY CLASS**

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
<u>Birchmeier v. Caribbean Cruise Line, Inc.</u> , 302 F.R.D. 240 (N.D. Ill. 2014)	<ul style="list-style-type: none"> District courts have flexibility to address manageability concerns. <u>See</u> Nichols Mem. at 27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiffs claimed that defendants violated the Telephone Consumer Protection Act of 1991 ("TCPA") by using prerecorded voice to call cellular and land lines. Low-value claims brought under TCPA. Defendants conceded there was a method for identifying at least a portion of the class using a list of telephone numbers that were called. No choice-of-law issues, because all claims arose under federal law. No questions of causation. Strict liability statute. No issue of comparative fault or intervening acts of third parties. Less complicated damages questions, because recovery limited to statutory damages (<u>i.e.</u>, \$500 or \$1,500).
<u>Buford v. H & R Block, Inc.</u> , 168 F.R.D. 340 (S.D. Ga. 1996)	<ul style="list-style-type: none"> Strong presumption against denying class certification based on manageability concerns. <u>See</u> Nichols Mem. at 27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiffs claimed that defendant tax preparer and banks falsely represented that a loan program was an actual tax refund. Court denied plaintiffs' motion for class certification. Held that individual issues of reliance predominated over common issues.

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
		<ul style="list-style-type: none"> • Low-value claims involving fees on tax-refund loans. • No choice-of-law issues, because all claims arose under federal law (<i>i.e.</i>, the National Bank Act, 12 U.S.C. §§ 85 and 86, and the Racketeer Influenced and Corrupt Organizations Act, 18 U.S.C. § 1964 (“RICO”)). • No issue of comparative fault or intervening acts of third parties. • Straightforward damages assessment, because damages consisted of fees incurred under loan program.
<u>Butler v. Sears, Roebuck and Co.</u> , 727 F.3d 796 (7th Cir. 2013)	<ul style="list-style-type: none"> • District courts can confine certification to liability questions and use individual hearings to determine the damages each class member sustained. <i>See</i> Nichols Mem. at 24, 27. 	<ul style="list-style-type: none"> • Not a personal injury case. Breach of warranty suit brought under the laws of six states. • Low-value claims involving allegedly defective washing machines. • Single defendant. • No issue of comparative fault or intervening acts of third parties. • Straightforward damages assessment -- the court noted that parties could likely agree on a schedule of damages based on the cost of fixing or replacing class members’ washing machines.

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
<u>Carnegie v. Household International, Inc.</u> , 376 F.3d 656 (7th Cir. 2004)	<ul style="list-style-type: none"> District courts must explore methods to overcome manageability problems. Individual damages hearings may be appropriate. <u>See</u> Nichols Mem. at 26-27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiff filed suit against defendant bank and tax preparer alleging RICO violation and breach of contract under the law of one state. Low-value claims involving fees incurred for tax-refund loans. No choice-of-law issues -- claims arose under federal law and law of a single state. No issue of comparative fault or intervening acts of third parties. Straightforward damages assessment, because damages were the fees actually incurred.
<u>In re Diet Drugs</u> , 2000 WL 1222042 (E.D. Pa. 2000), <u>aff'd</u> 369 F.3d 293 (3d Cir. 2004)	<ul style="list-style-type: none"> In her opinion certifying the NFL settlement class, Judge Brody noted that there are several examples of certified personal injury classes. <u>See</u> Nichols Mem. at 21 n. 23. 	<ul style="list-style-type: none"> Plaintiffs alleged cardiovascular problems stemming from taking two pharmacologically related diet pills. District court emphasized that “the class here involves a single defendant with essentially a single diet drug product.” <u>Diet Drugs</u>, 2000 WL 1222042, at *42. Settlement class so manageability concerns were not implicated. No issue of comparative fault or intervening acts of third parties. Experts agreed that there was a simple test that could determine if class members had compensable injuries, and there was no “latent” conditions. Settlement nullified individual issues of causation,

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
		injury and damages.
<p><u>In re IKO Roofing Shingle Products Liability Litigation</u>, 757 F.3d 599 (7th Cir. 2014)</p>	<ul style="list-style-type: none"> District courts can confine certification to liability questions and use individual hearings to determine the damages each class member sustained. <u>See</u> Nichols Mem. at 24, 27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiffs alleged defendant-roofing shingle manufacturer falsely told customers shingles met industry standard. Low-value claims involving defective roofing shingles. Less complicated questions of causation. No issue of comparative fault or intervening acts of third parties.
<p><u>In re Motor Vehicle Air Pollution Control Equipment</u>, 52 F.R.D. 398 (C.D. Cal. 1970)</p>	<ul style="list-style-type: none"> Court and class counsel must be imaginative in addressing manageability concerns. <u>See</u> Nichols Mem. at 27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiffs alleged defendants violated the Sherman Act, 15 U.S.C. § 1, by conspiring to impede the commerce of air pollution control equipment for motor vehicles. No choice-of-law issues, because claims arose under federal law. No issue of comparative fault or intervening acts of third parties.
<p><u>In re Oil Spill by the Oil Rig “Deepwater Horizon” in the Gulf of Mexico, on April 20, 2010</u>, 295 F.R.D. 112 (E.D. La. 2013)</p>	<ul style="list-style-type: none"> In her opinion certifying the NFL settlement class, Judge Brody noted that there are several examples of certified personal injury classes. <u>See</u> Nichols Mem. at 21 n. 23. 	<ul style="list-style-type: none"> Class certified for settlement only, so no determination that a trial of the class’s claims would be manageable. Settlement avoided need for inquiry into cause of claimant’s alleged injury. No choice of law issues as all legal questions arose under federal maritime law. Claims arose from single event -- explosion of the Deepwater Horizon -- and BP’s response during a “relatively short period of time in a tightly defined

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
		<p>geographic region.” 295 F.R.D. at 136.</p> <ul style="list-style-type: none"> • More than 17,000 short form joinders had been filed to date, some percentage of whom would sue individually absent settlement class certification; large-scale litigation of individual claims would represent misallocation of judicial resources.
<p><u>In re Phenylpropanolamine Products Liability Litigation</u>, 227 F.R.D. 553 (W.D. Wash. 2004)</p>	<ul style="list-style-type: none"> • In her opinion certifying the NFL settlement class, Judge Brody noted that there are several examples of certified personal injury classes. <u>See</u> Nichols Mem. at 21 n. 23. 	<ul style="list-style-type: none"> • Class certified for settlement only, so no assessment of manageability. • No choice of law issues. • No issue of comparative fault or intervening acts of third parties. • Settlement nullified individual issues of causation, injury and damages.
<p><u>In re Serzone Products Liability Litigation</u>, 231 F.R.D. 221 (S.D. W. Va. 2005)</p>	<ul style="list-style-type: none"> • In her opinion certifying the NFL settlement class, Judge Brody noted that there are several examples of certified personal injury classes. <u>See</u> Nichols Mem. at 21 n. 23. 	<ul style="list-style-type: none"> • Plaintiffs’ alleged injuries stemming from taking a certain antidepressant. • Class certified for settlement only, so no assessment of manageability. • No issue of comparative fault or intervening acts of third parties. • “This case involves one defendant, one product, and one course of conduct confined to a defined time period in which the defendant engaged in behavior that presented virtually identical risk to all the claimants.” 231 F.R.D. at 240. • Counsel agreed that there was “no risk of latent effects or future claims.” <u>Id.</u> at 228.

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
<u>Kartman v. State Farm Mutual Automobile Insurance Company</u> , 634 F.3d 883 (7th Cir. 2011)	<ul style="list-style-type: none"> District courts have flexibility to address manageability concerns. <u>See</u> Nichols Mem. at 27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiffs were Indiana homeowners whose homes were damaged by hail. Plaintiffs sued insurer for damages and injunction requiring insurer to re-inspect their roofs. Single defendant. District court refused to certify damages class under Fed. R. Civ. P. 23(b)(3) but certified class for injunctive relief under Fed. R. Civ. P. 23(b)(2). Seventh Circuit reversed certification of Rule 23(b)(2) class, because injunctive relief sought was neither appropriate nor final. No choice-of-law issues, because all claims arose under Indiana law. No issue of comparative fault or intervening acts of third parties.
<u>Mejdrech v. Met-Coil Systems Corp.</u> , 319 F.3d 910 (7th Cir. 2003)	<ul style="list-style-type: none"> Bifurcation of liability and damages determinations may be appropriate and efficient when there are issues common to class. <u>See</u> Nichols Mem. at 27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiffs alleged that storage tank on defendant's property leaked TCE, which contaminated the soil and groundwater beneath the class members' homes and reduced their property values. Single defendant. No choice of law issues, because all class members were residents of Illinois and were proceeding under the same state and federal laws. No issue of comparative fault or intervening acts of third parties.

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
<u>Mullen v. Treasure Chest Casino, LLC</u> , 186 F.3d 620 (5th Cir. 1999)	<ul style="list-style-type: none"> Personal injury classes are routinely certified. <u>See</u> Nichols Mem. at 29. 	<ul style="list-style-type: none"> Plaintiff crew members of defendant floating casino alleged respiratory illness caused by vessel's defective ventilation system. In affirming class certification, court emphasized lack of choice-of-law issues, because all claims arose under federal law (<u>i.e.</u>, the Jones Act). Putative class members all claimed injury from the very same allegedly defective ventilation system in the same facility over the same period of time. Court noted this distinguished the case from <u>Amchem</u>, where class members were exposed to asbestos from different sources over different time periods. Case was decided pre-<u>Dukes</u>. <u>See</u> <u>M.D. ex rel. Stukenberg v. Perry</u>, 675 F.3d 832, 839-40 (5th Cir. 2012) (the observation of the court in <u>Mullen</u> that "[t]he test for commonality is not demanding" abrogated by <u>Dukes</u>' requirement that class members suffer the same injury).
<u>Pella Corporation v. Saltzman</u> , 606 F.3d 391 (7th Cir. 2010)	<ul style="list-style-type: none"> Need for individual proof, without more, does not preclude class certification. <u>See</u> Nichols Mem. at 24. District courts can confine certification to liability questions and use individual hearings to determine the damages each class member sustained. <u>See id.</u> at 27. 	<ul style="list-style-type: none"> Not a personal injury case. Plaintiffs owned structures containing allegedly defective windows. Filed consumer fraud claims against manufacturer. Low-value claims involving allegedly defective windows. No complex choice of law issues -- district court certified subclasses for six states whose consumer protection statutes have nearly identical elements.

Case	Proposition(s) For Which Case Is Cited	Distinguishing Facts
		<ul style="list-style-type: none"> • Less complicated questions of causation -- claims largely turned on whether the product was defective. • No issue of comparative fault or intervening acts of third parties. • Straightforward damages assessment (<u>i.e.</u>, cost of replacing windows).
<u>Turner v. Murphy Oil USA, Inc.</u> , 234 F.R.D. 597 (E.D. La. 2006)	<ul style="list-style-type: none"> • Personal injury classes are routinely certified. <u>See</u> Nichols Mem. at 29. 	<ul style="list-style-type: none"> • Claims arose from a single incident. Plaintiffs alleged damages resulting from release of 25,110 barrels of crude oil from defendant's storage tank. • Single defendant. • Court certified claims arising under Louisiana law but refused to certify class as to common law tort claims due to choice-of-law issues. Court noted that "[b]oth this Court and the Fifth Circuit have refused to certify class actions in the past where the laws of multiple states are potentially applicable to the plaintiffs' claims, on the grounds that the claims are unmanageable and present too many issues requiring individual resolution." <u>Turner</u>, 234 F.R.D. at 608 (citing <u>Castano v. American Tobacco Company</u>, 84 F.3d 734, 741-43 (5th Cir. 1996); <u>In re Propulsid Products Liability Litigation</u>, 208 F.R.D. 133, 146-47 (E.D. La. 2002)). • No issue of comparative fault or intervening acts of third parties.

EXHIBIT H

EXHIBIT H
STATE LAW RULES GOVERNING DUTY AND CONTACT SPORTS

State	Rules Governing Duty and Contact Sports
Arkansas	Courts have not addressed contact sports exception.
Arizona	The Arizona Constitution provides that “[t]he defense of contributory negligence or assumption of risk, shall, in all cases, whatsoever, be a question of fact and shall, at all times, be left to the jury.” Ariz. Const. Art. 18, § 5. This provision prohibits adoption of a higher standard of care for sports participants. <u>See Estes v. Tripson</u> , 932 P.2d 1364, 1367 (Ariz. Ct. App. 1997) (applying traditional “reasonable care under the circumstances” standard to claim arising from collision that occurred during softball game).
California	Sports participants liable only for intentional misconduct or “conduct that is so reckless as to be totally outside the range of the ordinary activity involved in the sport.” <u>Knight v. Jewett</u> , 834 P.2d 696, 711 (Cal. 1992). Sports league is not insurer of athlete’s safety. League has duty to not increase risks to a participant over those inherent in the sport, but does not have a duty to decrease risks. <u>See West v. Sundown Little League of Stockton, Inc.</u> , 116 Cal.Rptr.2d 849, 854-55 (Cal. Ct. App. 2002) (affirming dismissal of complaint against local little league, coaches and league agents, and national little league organization filed by baseball player struck in the head by a fly ball).
Connecticut	Contact sports participants liable “only for reckless or intentional conduct and not for merely negligent conduct.” <u>Jaworski v. Kiernan</u> , 696 A.2d 332, 338-39 (Conn. 1997). Exception does not apply to golf or downhill skiing. <u>Id.</u> at 411-12 (explaining application of traditional negligence standard to claim arising from golf injury because golf is “neither a team sport . . . nor a sport where contact with other participants is part of the game”); <u>Jagger v. Mohawk Mountain Ski Area, Inc.</u> , 849 A.2d 813 (Conn. 2004) (contact sports exception does not apply to downhill skiing).
Illinois	Participants in contact sports owe a duty to refrain from willful and wanton or intentional misconduct but are not liable for simple negligence. <u>See Pfister v. Shusta</u> , 657 N.E.2d 1013, 1017-18 (Ill. 1995). Contact sports exception applies to organizational defendants. Such defendants are only liable if they acted with intent to cause injury or engaged in conduct “totally outside the range of the ordinary activity.” <u>Karas v. Strevell</u> , 884 N.E.2d 122, 136-37 (Ill. 2008) (case involving claims that hockey associations failed to adequately enforce rule against bodychecking from behind). <u>But see Gvillo v. DeCamp Junction, Inc.</u> , 959 N.E.2d 215 (Ill. App. Ct. 2011) (distinguishing <u>Karas</u> and holding contact sports exception did not apply where defendant organization allegedly acted negligently in setting up a softball field). Contact sports exception does not apply to golf or downhill skiing. <u>See Zurla v. Hydel</u> , 681 N.E.2d 148, 152 (Ill. App. Ct. 1997) (golf); <u>Novak v. Virene</u> , 586 N.E.2d 578, 580 (Ill. App. Ct. 1991) (downhill skiing).

State	Rules Governing Duty and Contact Sports
Indiana	Traditional negligence framework -- reasonableness under the circumstances -- applies to conduct of sports participants. <u>See Pfenning v. Lineman</u> , 947 N.E.2d 392, 403 (Ind. 2011).
Iowa	Participants in contact sports owe duty to refrain from recklessness and intentional misconduct. <u>See Leonard ex rel. Meyer v. Behrens</u> , 601 N.W.2d 76, 81 (Iowa 1999) (applying contact sports exception to paintball).
Maine	Courts have not addressed contact sports exception.
Maryland	Voluntary participants in sporting events assume the risks inherent to that activity, including the risk that other participants may be negligent; sports injury claimant must allege reckless or intentional misconduct. <u>See Am. Powerlifting Ass'n v. Cotillo</u> , 934 A.2d 27, 35 (Md. 2007).
Massachusetts	Sports participants owe duty to refrain from reckless misconduct. <u>See Gauvin v. Clark</u> , 537 N.E.2d 94, 97 (Mass. 1989). Reckless misconduct standard applies to non-contact sports such as golf. <u>See Gray v. Giroux</u> , 730 N.E.2d 338, 341 (Mass. App. Ct. 2000).
Missouri	“Cause of action for personal injuries incurred during athletic competition must be predicated on recklessness, not mere negligence.” <u>Ross v. Clouser</u> , 637 S.W.2d 11, 13-14 (Mo. 1982) (en banc).
Nebraska	Participant in contact sport liable for injuries only if her conduct is willful or reckless. <u>See Dotzler v. Tuttle</u> , 449 N.W.2d 774, 779 (Neb. 1990).
Nevada	Traditional negligence standard applies to sports injury cases. <u>See Auckenthaler v. Grundmeyer</u> , 877 P.2d 1039, 1043-44 (Nev. 1994).
New Hampshire	Sports participants, sponsors and organizers are not liable for negligent (or even reckless or intentional conduct) that is not outside the range of ordinary activity involved in the sport. <u>See Allen v. Dover Co-Recreational Softball League</u> , 807 A.2d 1274, 1285 (N.H. 2002). Associations have “duty to promulgate or enforce rules that minimize the risk of injury” only if “failing to promulgate and enforce such rules created risks outside the risks ordinarily involved in” the sport. <u>Id.</u> at 1288.
New Jersey	Sports participants owe a duty to avoid infliction of injury caused by reckless or intentional conduct. <u>See Crown v. Campo</u> , 643 A.2d 600, 607 (N.J. 1994). Reckless or intentional conduct standard of care applies generally to recreational sports, including non-contact sports like golf. <u>Schick v. Ferolito</u> , 767 A.2d 962 (N.J. 2001).
New Mexico	Reckless or willful misconduct standard of care applies to contact sports participants. <u>See Kabella v. Bouschelle</u> , 672 P.2d 290, 294 (N.M. Ct. App. 1983).
Ohio	Sports participants assume the ordinary risks of the sport and cannot recover for any injury unless they can prove the injury was caused by reckless or intentional misconduct. <u>See Marchetti v. Kalish</u> , 559 N.E.2d 699, 703-04 (Ohio 1990).

State	Rules Governing Duty and Contact Sports
Pennsylvania	Sports associations have no duty to protect participants from risks inherent to the sports. <u>See Craig v. Amateur Softball Ass'n of Am.</u> , 951 A.2d 372, 375-76 (Pa. Super. Ct. 2008).
Rhode Island	Recklessness or willful misconduct standard of care applies to sports participants. <u>See Kiley v. Patterson</u> , 763 A.2d 583, 586 (R.I. 2000).
Wisconsin	State statute provides immunity from negligence actions for participants in recreational activity that involves physical contact between participants. <u>See Noffke v. Bakke</u> , 760 N.W.2d 156, 166 (Wis. 2009) (holding statute applies to cheerleading).

EXHIBIT I

EXHIBIT I
SAMPLE PATTERN JURY INSTRUCTIONS

State	Negligence Definition	Proximate Cause	Contributory/ Comparative Negligence
California	<p>“Negligence is the doing of something which a reasonably prudent person would not do, or the failure to do something which a reasonably prudent person would do, under circumstances similar to those shown by the evidence. It is the failure to use ordinary or reasonable care. Ordinary or reasonable care is that care which persons of ordinary prudence would use in order to avoid injury to themselves or others under circumstances similar to those shown by the evidence.”</p> <p style="text-align: right;">Cal. Civil Jury Instructions 3.10</p>	<p>“The law defines cause in its own particular way. A cause of injury/damage/loss/harm is something that is a substantial factor in bringing about an injury/damage/loss/harm.”</p> <p style="text-align: right;">Cal. Civil Jury Instructions 3.76</p>	<p>“Contributory negligence is negligence on the part of a plaintiff which, combining with the negligence of a defendant, contributes as a cause in bringing about the injury.</p> <p>Contributory negligence, if any, on the part of the plaintiff does not bar a recovery by the plaintiff against the defendant but the total amount of damages to which the plaintiff would otherwise be entitled must be reduced in proportion to the amount of negligence attributable to the plaintiff.”</p> <p style="text-align: right;">Cal. Civil Jury Instructions 3.50</p>
Delaware	<p>“This case involves claims of negligence. Negligence is the lack of ordinary care; that is, the absence of the kind of care a reasonably prudent and careful person would exercise in similar circumstances. That standard is your guide. If a person’s conduct in a given circumstance doesn’t measure up to the conduct of an ordinarily prudent and careful person, then that person was negligent. On the other hand, if the person’s conduct does measure up to the conduct of a reasonably prudent and careful person, the person wasn’t negligent.”</p> <p style="text-align: right;">Del. Pattern Jury Instructions - Civil § 5.1</p>	<p>“A party’s negligence, by itself, is not enough to impose legal responsibility on that party. Something more is needed: the party’s negligence must be shown by a preponderance of the evidence to be a proximate cause of the [accident / injury]. Proximate cause is a cause that directly produces the harm, and but for which the harm would not have occurred. A proximate cause brings about, or helps to bring about, the [accident / injury], and it must have been necessary to the result.”</p> <p style="text-align: right;">Del. Pattern Jury Instructions - Civil § 21.1</p>	<p>“[Defendant’s name] alleges that [plaintiff’s name]’s negligence proximately caused the accident. Negligence is negligence no matter who commits it. When the plaintiff is negligent, we call it contributory negligence. Under Delaware law, a plaintiff’s contributory negligence doesn’t mean that the plaintiff can’t recover damages from the defendant as long as the plaintiff’s negligence was no greater than the defendant’s negligence. Instead of preventing a recovery, Delaware law reduces the plaintiff’s recovery in proportion to the plaintiff’s negligence.</p> <p>If you find contributory negligence was a proximate cause of the [accident / injury], you must determine the degree of that negligence, expressed as a percentage, attributable to [plaintiff’s name]. Using 100% as the total combined negligence of the parties, you must determine what percentage of negligence is attributable to</p>

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			<p>[plaintiff's name]. I will furnish you with a special-verdict form for this purpose. If you find that [plaintiff's name]'s negligence is no more than half the total negligence, I will reduce the total amount of [plaintiff's name]'s damages by the percentage of [his/her] contributory negligence. If you find that [plaintiff's name]'s negligence is more than half the total negligence, [plaintiff's name] may not recover any damages."</p> <p>Del. Pattern Jury Instructions - Civil § 5.12</p>
Illinois	<p>"When I use the word 'negligence' in these instructions, I mean the failure to do something which a reasonably careful person would do, or the doing of something which a reasonably careful person would not, under circumstances similar to those shown by the evidence. The law does not say how a reasonably careful person would act under those circumstances. That is for you to decide."</p> <p>Ill. Pattern Jury Instructions - Civil 10.01</p> <p>"If you decide there is evidence tending to show that the [decedent / plaintiff / defendant] was a person of careful habits, you may infer that he was in the exercise of ordinary care for his own safety [and for the safety of others] at and before the time of the occurrence, unless the inference is overcome by other evidence. In deciding the issue of the exercise of ordinary care by the decedent/plaintiff/ defendant you may consider this inference and any other evidence upon the subject of the decedent's/plaintiff's/defendant's care."</p> <p>Ill. Pattern Jury Instructions - Civil 10.08</p>	<p>"When I use the expression 'proximate cause,' I mean a cause that, in the natural or ordinary course of events, produced the plaintiff's injury. It need not be the only cause, nor the last or nearest cause. It is sufficient if it combines with another cause resulting in the injury."</p> <p>Ill. Pattern Jury Instructions - Civil 15.01</p>	<p>"When I use the expression 'contributory negligence,' I mean negligence on the part of the plaintiff that proximately contributed to cause the [alleged] injury/death/property damage."</p> <p>Ill. Pattern Jury Instructions - Civil 11.01</p>

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Massachusetts	<p>“The law defines negligence as the failure of a person to exercise that degree of care which a reasonable person would exercise in the circumstances. Negligence is doing something that a reasonably prudent person in the ordinary course of human events would not do, or failing to do something that a reasonable person of ordinary prudence would do. The classic definition of negligence is this: Negligence is the failure of a responsible person, either by omission or by action, to exercise that degree of care, diligence, and forethought which, in the discharge of the duty then resting on [him/her], the person of ordinary caution and prudence ought to exercise under the particular circumstances. It is a want of diligence commensurate with the requirement of the duty at the moment imposed by the law.</p> <p>Negligence is the performance or the omission of some act in violation of a legal duty. Ordinarily, where a duty of care is established by law, the standard by which a party’s performance is measured is the conduct expected of a reasonably prudent person in similar circumstances. The standard is not established by the most prudent person conceivable, nor by the least prudent, but by the person who is thought to be ordinarily prudent. The same standard is frequently expressed in terms of ‘reasonable care.’”</p> <p>Mass. Super. Ct. Civil Practice Jury Instructions § 2.1.2</p>	<p>“On the causation issue, there is one other consideration you must address. A defendant’s liability is limited to those harms that result from the risks that made the defendant’s conduct negligent. You must decide whether the harm to the plaintiff is within the scope of the defendant’s liability. To do that, you must first consider why you found the defendant negligent. You should consider all of the dangers that the defendant should have taken reasonable steps to avoid. The defendant is liable for the plaintiff’s harm if you find that the plaintiff’s harm arose from the same general type of danger that was one of those that the defendant should have taken reasonable steps to avoid. If the plaintiff’s harm, however, did not arise from the same general dangers that the defendant failed to take reasonable steps to avoid, then you must find that the defendant is not liable for the plaintiff’s harm.”</p> <p>Mass. Super. Ct. Civil Practice Jury Instructions § 2.1.8(d)</p>	<p>“If the negligence of the plaintiff is equal to or less than the negligence of all the defendants combined, the plaintiff’s damages will be diminished by the percentage of [his/her] own negligence. If the negligence of the plaintiff is less than 50 percent, the amount you determine to be the plaintiff’s damages will be reduced by that percentile by the clerk. For example, if you determine that the plaintiff was 20 percent negligent and that the combined negligence of all defendants was 80 percent, the amount of the plaintiff’s total damages will be reduced by 20 percent by the clerk. You should write in the full amount of the damages without making any deduction for comparative negligence, keeping in mind that the clerk will make any such deduction after you render your verdict. If you find that the negligence of the plaintiff is greater than the combined negligence of the defendants, do not answer the damages question.”</p> <p>Mass. Super. Ct. Civil Practice Jury Instructions § 2.1.11</p>

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North Carolina	<p>“Negligence refers to a person’s failure to follow a duty of conduct imposed by law. Every person is under a duty to use ordinary care to protect himself and others from injury/damage.</p> <p>Ordinary care means that degree of care which a reasonable and prudent person would use under the same or similar circumstances to protect himself and others from injury/damage. A person’s failure to use ordinary care is negligence.”</p> <p>N.C. Pattern Instructions - Civil 102.11</p>	<p>“The plaintiff not only has the burden of proving negligence, but also that such negligence was a proximate cause of the injury/damage.</p> <p>Proximate cause is a cause which in a natural and continuous sequence produces a person’s injury/damage, and is a cause which a reasonable and prudent person could have foreseen would probably produce such injury/damage or some similar injurious result.”</p> <p>N.C. Pattern Instructions - Civil 102.19</p>	<p>“The test of what is negligence, as I have already defined it, is the same for the plaintiff as for the defendant. If the plaintiff’s negligence joins with the negligence of the defendant in proximately causing the plaintiff’s own injury/damage, it is called contributory negligence, and the plaintiff cannot recover.”</p> <p>N.C. Pattern Instructions - Civil 104.10</p>
Oregon	<p>“The law requires every person to use reasonable care to avoid harming others. A person’s conduct is negligent if that person fails to use reasonable care. Reasonable care is the degree of care and judgment used by reasonably careful people in the management of their own affairs to avoid harming themselves or others. A person fails to use reasonable care when that person does something that a reasonably careful person would not do, or fails to do something that a reasonably careful person would do under similar circumstances.</p> <p>In deciding whether a person used reasonable care, consider the dangers apparent or reasonably foreseeable when the events occurred. Do not judge the person’s conduct in light of later events; instead, consider what the person knew or should have known at the time.”</p> <p>Or. Uniform Civil Jury Instructions 20.02</p>	<p>“Many factors or things may operate either independently or together to cause harm / injury. In such a case, each may be a cause of the harm / injury even though the others by themselves would have been sufficient to cause the same harm / injury. If you find that the defendant’s act or omission was a substantial factor in causing the harm / injury to the plaintiff, you may find that the defendant’s conduct caused the harm / injury even though it was not the only cause.”</p> <p>Or. Uniform Civil Jury Instructions 23.02</p>	<p>“The plaintiff and the defendant have each alleged that the damage was caused by the other’s fault / negligence. If you find that both the defendant and the plaintiff were at fault / negligent and that their fault / negligence caused the alleged damage, then you must compare the fault / negligence of the plaintiff to the [fault / negligence of the defendant.</p> <p>In making this comparison, you must measure the percentage of fault / negligence of each and not the percentage of damage caused by each.</p> <p>The comparison of fault / negligence must be expressed in terms of percentages that total 100 percent. If the plaintiff’s fault / negligence is more than 50 percent, then your verdict is for the defendant. On the other hand, if the plaintiff’s fault / negligence is 50 percent or less, then your verdict is for the plaintiff.</p> <p>Do not reduce the amount of the plaintiff’s damages, if any, as a result of your comparison. I will reduce the amount of</p>

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			your verdict by the percentage of the plaintiff's fault / negligence, if any. Or. Uniform Civil Jury Instructions 21.02
South Carolina	<p>“What is negligence? Negligence is defined in the law as the absence of due care. The want or lack of due care or ordinary care. The word carelessness conveys the same idea as negligence. Those two terms are synonymous. Negligence is the breach of a duty of care owed to the plaintiff by the defendant. Negligence is the failure, by omission or commission, to exercise due care as a person of ordinary reason and prudence would exercise in the same circumstances. It is the doing of some act which a person of ordinary prudence would not have done under similar circumstances or failure to do what a person of ordinary prudence would have done under similar circumstances.</p> <p>In determining whether a particular act is negligent, the test you apply is what would a person of ordinary reason and prudence do under those circumstances at that time and place.”</p> <p style="text-align: right;">Anderson's S.C. Requests to Charge - Civil § 20-1</p>	<p>“The touchstone of proximate cause in South Carolina is foreseeability. That is, foreseeability of some injury from a negligent act or omission is a prerequisite to its being a proximate cause of the injury for which recovery is sought. The test of foreseeability is whether some injury to another is the natural and probable consequence of the complained of act. The defendant may be held liable for anything which appears to have been a natural and probable consequence of his negligence. The law only requires reasonable foresight . . . Proximate cause does not mean the sole cause. The defendant's conduct can be a proximate cause if it was at least one of the direct, concurring causes of the injury. The law defines proximate cause of an injury to be something that produces a natural chain of events which, in the end, brings about the injury. In other words, proximate cause is the direct cause, without which the injury would not have occurred. If the accident would have happened as a natural and probable consequence, even in the absence of the alleged breach, then the plaintiff has failed to demonstrate proximate cause.”</p> <p style="text-align: right;">Anderson's S.C. Requests to Charge - Civil § 20-2</p>	<p>“Comparative negligence is the law in South Carolina. Under the doctrine of comparative negligence, the plaintiff's negligence does not automatically bar recovery unless such negligence exceeds that of the defendant. A plaintiff in a negligence action may recover damages if his negligence is not greater than that of the defendant. The amount of the plaintiff's recovery shall be reduced in proportion to the amount of his negligence.</p> <p>You, the jury, must apportion fault between the plaintiff and defendant in a negligence action. The plaintiff may recover damages when his negligence is not greater than that of the defendant. The plaintiff's damages, however, are reduced in proportion to the amount of his negligence.”</p> <p style="text-align: right;">Anderson's S.C. Requests to Charge - Civil § 23-1</p>

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Tennessee	<p>“Negligence is the failure to use ordinary or reasonable care. It is either doing something that a reasonably careful person would not do, or the failure to do something that a reasonably careful person would do, under all of the circumstances in this case. The mere happening of an injury or accident does not, in and of itself, prove negligence.</p> <p>A person may assume that every other person will use reasonable care, unless a reasonably careful person has cause for thinking otherwise.”</p> <p>Tenn. Pattern Instructions - Civil 3.05</p>	<p>“Once you have determined that a defendant’s negligence is a cause in fact of the plaintiff’s injury, you must decide whether the defendant’s negligence was also a legal cause of the plaintiff’s injury.</p> <p>The law in Tennessee sets out two requirements to determine whether an act or omission was a legal cause of the injury or damage.</p> <ol style="list-style-type: none"> 1. The conduct must have been a substantial factor in bringing about the harm being complained of; and, 2. The harm giving rise to the action could have been reasonably foreseen or anticipated by a person of ordinary intelligence and care. <p>To be a legal cause of an injury there is no requirement that the cause be the only cause, the last act, or the one the nearest to the injury, so long as it is a substantial factor in producing the injury or damage.</p> <p>The foreseeability requirement does not require the person guilty of negligence to foresee the exact manner in which the injury takes place or the exact person who would be injured. It is enough that the person guilty of negligence could foresee, or through the use of reasonable care, should have foreseen the general manner in which the injury or damage occurred.”</p> <p>Tenn. Pattern Instructions - Civil 3.22</p>	<p>“If you find that a party was negligent and that the negligence was a cause in fact and also a legal cause of the injury or damages for which a claim was made, you have found that party to be at fault. . . . It is my responsibility under the law to reduce the amount of damages you assess against any party by the percentage of fault, if any, that you assign to that party. A party claiming damages will be entitled to damages if that party’s fault is less than 50% of the total fault in the case. A party claiming damages who is 50% or more at fault, however, is not entitled to recover any damages whatsoever.”</p> <p>Tenn. Pattern Instructions - Civil 3.50</p>

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Virginia	<p>“The law does not require a person to know that he is absolutely safe before taking a given course of action. He is only required to exercise ordinary care—such care as a reasonably prudent person would exercise under the circumstances.”</p> <p>Va. Practice Jury Instructions § 12:1</p>	<p>“A proximate cause of an injury, accident, or damage is a cause which in the natural and continuous sequence produces the accident, injury, or damage. It is a cause without which the accident, injury, or damage would not have occurred.”</p> <p>Va. Practice Jury Instructions § 12:15</p>	<p>“If the jury believes from the evidence that the defendant was negligent and that such negligence was a proximate cause of the collision, and if you further believe from the evidence that the plaintiff was also negligent and that such negligence proximately contributed to cause the collision, then your verdict shall be in favor of the defendant. The law does not undertake to apportion or balance the negligence of the parties where both are at fault in order to ascertain which one is most at fault, but the plaintiff is barred from recovery if he was guilty of any negligence that proximately contributed to cause the collision.”</p> <p>Va. Practice Jury Instructions § 12:6</p>